hypothesis – now fully accepted throughout the cognitive neuroscience community – that the cerebellum is centrally involved in linguistic acquisition. Since Professor Bishop's research area is specific language impairment, she must have repressed this knowledge.

Bishop's assertion that the cerebellar-based explanation is invalidated because dyslexics do not show motor or balance problems perpetuates a pernicious myth – that the cerebellar deficit hypothesis claims the underlying cause of reading difficulties in dyslexia is some lack of motor skill. This is simply untrue. The cerebellum contains over half the cells in the brain. Different parts of it are involved in the acquisition of language and motor fluency. The cerebellar deficit hypothesis[4] states that the three primary routes to reading difficulties arise from weak phonology, weak verbal working memory and inefficient skill automatisation; all of which are attributed to cerebellar deficit. Presence of motor skill deficits is not claimed to be a cause of reading difficulties.

The statement that we ignored the results of the control group is bizarre, since we published these control data. Bishop criticises our use of Nicolson's Dyslexia Screening Test (DST) because it is not a 'sensitive measure of individual differences', but this is exactly what it has been designed to do, and which is why it has become the 'industry standard' measure, with established high test–retest reliability.

Bishop further asserts that the gains shown for our sample are practice effects. It is absurd to suggest that a minute or two of experiencing tests of reading, writing, rapid naming, etc., in which fluency is a major dimension, is likely to lead to an improvement 6 months later!

Bishop criticises our use of the Standard Assessment Tests (SATs). They are valid, reliable and up-to-date measures that have been developed to test acquisition of the curriculum in British schools, and are the currency by which the educational system judges itself.

A final criticism is the vestibular and postural tests used at Dore Achievement Centres that we also reported, where it is argued that we did not possess norms for children on these tests. This is true, but lack of norms does not indicate any necessary lack of objectivity. Most clinical tests are based on clinical judgements by professionals. Dore's tests are objective with automatically generated evidence.

Bishop talks of a 'Miracle Cure' that we have never done. She asserts the data show that the 'intervention improves nothing other than the skills trained in the exercises' – this is a travesty. Our work shows that there were significant gains in physical co-ordination following treatment (maintained post-treatment) and that there were significant and maintained gains in mental co-ordination and attention (ignored by Bishop), as measured by phonological skills, verbal working memory and processing speed. The literacy effects were more mixed, with scores on the reading test 'with good psychometric properties' and SATs improving but those on the DST not doing so, a complex set of findings that were extensively discussed in the paper but ignored by Bishop.

Controversy in science is its lifeblood, as invalid research and judgements are exposed, and the valid subsequently form the bedrock of further work. We would only want to encourage discussion of our work.

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20 February 2008

Dear Editor,

SUCCESSFUL OUTCOME OF FAMILY THERAPY FOR ANOREXIA NERVOSA?

We were surprised to read about 'an impressive recovery' from anorexia nervosa in a case report of a patient who was treated with the Maudsley family therapy approach.[1] The accompanying letter describes a less favourable outcome of the same treatment in another anorexic patient.

The development of medical science was recently described by Richard Smith, who served as Editor of the British Medical Journal for 13 years.[2] Smith notes that 'The history of medicine is largely a history of ineffective treatments. "The role of the doctor," joked Voltaire, "is to amuse the patient while nature takes its course" . . . The simplest way to see if a treatment works is to give it to a patient and see what happens. The resulting publication is called a case report and was for many years the mainstay of medical journals. You don't need to be much of a scientist to see the limitations of such evidence. What would have happened if the patient hadn't been given the treatment? How do I know as a reader whether or not the reported patient is like my patient and whether or not what happened to the patient would happen to my patient?' Smith also discusses many other problems with the case report.[2]

The randomised controlled trial (RCT) is now the accepted form, or rather, the 'gold standard' for the development of medical treatments. Smith even refers to the RCT as 'one of the greatest scientific inventions of the 20th century'.[2]

We ourselves published the successful outcome of the treatment of an individual Australian patient as a case report in this journal.[3] However, this was after the treatment had been demonstrated, in an RCT, to be effective.[4] We want to make the treatment available to Australian and other patients, and in an

initial effort to do so, we chose to describe the successful outcome in our first Australian patient in a case report. However, as we have recently pointed out in a report of the outcome in a larger group of Australian patients, our treatment should be compared with the treatments used in Australia in an RCT.[5]

If, on the other hand, a treatment has been demonstrated to be minimally effective in RCTs, it should no longer be used. We recently reviewed the RCTs which have evaluated the effect of family-based treatments for anorexia nervosa and found no compelling evidence that family therapy is effective.[6]

If an ineffective treatment is still used, as is often the case in the management of anorexia nervosa, and a patient improves while receiving that treatment, one should not only be surprised; one should also conclude that the improvement was probably not because of the treatment. However, as Peter Medawar pointed out, 'If a person is poorly, receives treatment to make him better, and then gets better, then no power of reasoning known to medical science can convince him that it may not have been the treatment that restored his health'.[7]

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20 February 2008

Dear Editor,

As parents of a child who suffered from anorexia for over 4 years, we read with interest the case report 'Anorexia, Maudsley and an impressive recovery: One family's story'.[1]

The authors of this report clearly had success using the Maudsley approach in treating their daughter's anorexia, and we congratulate them for this.

However, in our view, the report raises some questions about the Maudsley treatment that require serious consideration, given that this form of treatment appears to be fast becoming recognised as a 'treatment of choice' in Australia.[2]

It appears from the report that the patient in question was aged 15 at the time of treatment. But what of patients who are in their late teens, or early adulthood? It is never easy to get an anorexic to eat, but surely there is more chance of this happening when the patient is still relatively young and more likely to be compliant with parental wishes. We had some success with the Maudsley treatment when our daughter was 13. However, when she relapsed in her late teens, we found it impossible to exert the level of control over her eating that was necessary to subvert the illness.

The report skirts around the impact of the Maudsley treatment on other family members. What is the effect of the episodes of 'physical and verbal aggression' and the 'fighting and upset' at mealtimes on younger siblings? We have three other children, all of whom developed anxiety symptoms as a result of being exposed – for many months – to their sister's severe distress at mealtimes. They have all since told us that the period during which we were using Maudsley therapy was the worst time of their lives. Our 10-year-old witnessed us untying a noose from his sister's neck after she attempted to hang herself in her bedroom. No child should ever be subjected to that level of trauma in their own home.

We note that one of the authors of the case report had to give up work for the duration of her daughter's illness and that this was accepted as 'an inevitable consequence of having a child with a debilitating illness'. But what if family finances are such that this is not possible? What of single-parent families?

The rate of relapse after family therapy is yet to be satisfactorily explained.[3] We were not warned of this possibility and were shocked by our daughter's severe relapse, less than 18 months after her supposed 'recovery'.

Finally, what options are available for Australian patients when the Maudsley treatment fails? The options offered to our daughter included long-term admission to an adult psychiatric hospital, high doses of antidepressants and antipsychotics and years of psychotherapy to find the 'reason' for her anorexia.

None of these options were acceptable to her, or to us. Instead, we took her to the Karolinska Institute in Stockholm, Sweden where she was treated for 5 months using the Mandometer model of treatment. This form of treatment seeks to achieve weight restoration through the use of the Mandometer, a computerised biofeedback device that monitors eating rate and satiety.[4] In our daughter's case, it also involved the use of heat therapy to reduce the overwhelming urge to exercise that was a key feature of her anorexia. She was also assigned an individual case worker who had responsibility for monitoring her food intake and supporting her through difficult meals. This freed us up to simply be her parents again, rather than exhausted 'food police'.

Since her return to Australia, our daughter has resumed school and a normal teenage life. She has maintained a healthy weight and she no longer exhibits disordered eating behaviour or psychiatric symptoms.

The Maudsley model has obvious attractions for governments because it shifts the cost and responsibility of treatment away

from the public health system, placing it instead on the shoulders of the family of the anorexia sufferer. But for some families, the costs and responsibilities are too great. Sometimes, weight restoration and recovery cannot take place within the family environment, no matter how hard the family tries.

The Mandometer treatment provides a clinically proven, effective form of treatment for anorexia.[5] As such, it should be accessible and affordable for all Australian patients. Unfortunately, however, Mandometer treatment currently receives no funding from the Australian government and seriously ill patients must travel overseas for treatment.

This situation is unlikely to change until the Mandometer treatment is able to be compared in randomised controlled trials with Maudsley and other forms of treatment used in Australia. The Mandometer Clinic has expressed a willingness to participate in such a trial.[6] However, we understand that the Australian medical establishment has not shown any real interest in pursuing this avenue. Why not?

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31 January 2008

Dear Editor,

TRICHOBEZOAR IN A CHILD WITH UNDIAGNOSED COELIAC DISEASE

We write with an interesting case that we have recently encountered, which highlights the unusual ways in which children can present with Coeliac disease.

In this case, Coeliac disease presented with alopecia and iron deficiency anaemia. The anaemia was initially thought to be secondary to mal-absorption caused by trichobezoar. However, the iron deficiency state may have been a primary phenomenon due to underlying Coeliac disease, causing pica. In this instance, pica manifested itself as trichotillomania and trichobezoar. To our knowledge, this is the first described presentation of Coeliac disease in this fashion.

A 5-year-old girl initially presented to the dermatologist with a 6-week history of hair loss. She was clinically pale with a hypochromic microcytic anaemia and was referred to paediatric gastroenterology. For the previous 18 months, she had complained of intermittent abdominal pain, post-prandial vomiting, pale stools, decreased appetite and subsequent failure to thrive were reported. There was no prior medical or psychiatric history and no relevant family history.

She weighed 16 kg (9th centile for age) with pallor and non-scarring alopecia, worst at the vertex with absence of telogen. Abdominal examination revealed a mass in the left upper quadrant. The mother recalled prior observation of hair ingestion at this point. Abdominal X-ray revealed an opacity in the left upper quadrant.

Endoscopy confirmed the diagnosis of trichobezoar and duodenal biopsies were obtained to exclude Coeliac disease. The trichobezoar was too large to snare and remove endoscopically and thus, a mini-laparotomy and gastrotomy were performed to remove the bezoar (Fig. 1).

Her duodenal biopsies demonstrated changes consistent with gluten sensitive enteropathy of villous atrophy and crypt hyperplasia. The histological findings were supported by positive tissue IgA transglutaminase and anti-endomysial IgA antibodies. Treatment with gluten-free diet resulted in resolution of anaemia, improvement of weight gain and appetite.

Several factors in this case were not consistent with a simple diagnosis of iron deficiency secondary to gastric trichobezoar. Of note, her gastrointestinal symptoms that could have been caused by underlying Coeliac disease had been present for 18 months. Alopecia and ingestion of hair had only been noted in the final 6 weeks before presentation.

We, therefore, postulate the following sequence of events: Coeliac disease malabsorption leading to iron deficiency anaemia and resulting pica, in this case trichotillomania.

Gastric trichobezoars are uncommon in childhood and generally result from trichotillomania, the compulsion to eat hair. Trichobezoar is associated with iron deficiency anaemia[1], which often results in pica (the compulsion to ingest non-nutritious substances)[2] The most commonly ingested substance is ice;

Fig. 1 Trichobezoar retrieved at mini-laparotomy.

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