An Audit of the Thyroid Screening Programme in the Peninsula Maternal and Neonatal Services

by

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DECLARATION

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ABSTRACT

Objective
To audit the cord blood thyroid screening programme in the Peninsula Maternal and Neonatal Services (PMNS) in the 5 year period from 01/01/2000 to 31/12/2004, focusing on coverage, recall rate and success, number of cases detected, incidence of congenital hypothyroidism in this population; and cost efficiency of the programme.

Method
All babies born in the PMNS from 01/01/2000 to 31/12/2004 were included in the audit. The medical records of all babies recalled following an abnormal screen were examined. 140 507 babies were born in the PMNS during the audit period, while 130 389 primary Thyroid Stimulating Hormone (TSH) screens were done (92.8% coverage). 2 207 of the screened babies had abnormal results requiring review.

Main outcome measures
Result of review TSH and free Thyroxine (fT4), age at time of first review, cause of hypothyroidism, age at starting therapy, and the cost of the screen.

Results
13 cases of congenital hypothyroidism were detected out of the 751 abnormal screened individuals that were reviewed. 6 cases have developmental abnormalities of the thyroid (46%), 2 have dyshormonogenesis (15%), 1 is a probable uptake defect (7.7%), 1 was as a result of maternal Graves disease, which resolved after 6 months (7.7%), and in 3 cases the cause was undiagnosed because they were lost to follow up. The average age at review was 62 days.
The corrected incidence is calculated at 1: 3 448, which compares with the published general population.
The cost of the programme was R620 348.30 for 2004, which equates to R22.02 per screened baby, or R221 552.96 per actual detected case of congenital hypothyroidism.
Conclusion
The current screening programme reveals major problems in the recall success, with only 34% of abnormal screens returning for review, and a default rate of 31% in those detected as having congenital hypothyroidism. The greatest contributing factor is that there is no education, neither population nor maternal, about the screening programme and/or hypothyroidism.

Despite these problems, this audit identified an incidence that emphasizes the importance of this screening programme; and reflects its financial viability.

Recommendations
The greatest contribution to improving the success of the screening programme will be education. The delivery unit staff need updated information about the programme and its coverage; and the mothers need education about the screening for congenital hypothyroidism, starting in the antenatal clinics and continuing through to the well baby clinics.

Dedicated staff need to process the screening data more efficiently, and an improved recall system needs to be implemented. The recall rate can be decreased by changing cut-off TSH levels.

A protocol for recall, review and follow up must be constructed and piloted to achieve established standards of care.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Title page</td>
<td>i</td>
</tr>
<tr>
<td>Declaration</td>
<td>ii</td>
</tr>
<tr>
<td>Abstract</td>
<td>iii</td>
</tr>
<tr>
<td>Table of contents</td>
<td>v</td>
</tr>
<tr>
<td>Abbreviations</td>
<td>viii</td>
</tr>
<tr>
<td>1. Introduction</td>
<td>1</td>
</tr>
<tr>
<td>1.1 Congenital hypothyroidism and the history of screening neonates</td>
<td>2</td>
</tr>
<tr>
<td>1.2 Materials and methods of screening in the PMNS</td>
<td>4</td>
</tr>
<tr>
<td>2. Study design and research method</td>
<td>6</td>
</tr>
<tr>
<td>3. Results</td>
<td>7</td>
</tr>
<tr>
<td>3.1 Coverage</td>
<td>7</td>
</tr>
<tr>
<td>3.2 Secondary screening</td>
<td>7</td>
</tr>
<tr>
<td>3.3 Screen results</td>
<td>7</td>
</tr>
<tr>
<td>3.4 Gender ratio</td>
<td>9</td>
</tr>
<tr>
<td>3.5 Diagnoses</td>
<td>9</td>
</tr>
<tr>
<td>3.6 Treatment and follow up</td>
<td>9</td>
</tr>
<tr>
<td>3.7 Neurodevelopmental outcome</td>
<td>9</td>
</tr>
<tr>
<td>3.8 Cost of the screening programme</td>
<td>10</td>
</tr>
<tr>
<td>3.8.1 Consumables</td>
<td>10</td>
</tr>
<tr>
<td>3.8.2 Staff</td>
<td>10</td>
</tr>
<tr>
<td>3.8.3 Equipment</td>
<td>11</td>
</tr>
<tr>
<td>3.8.4 Additional tests</td>
<td>12</td>
</tr>
<tr>
<td>3.8.5 Treatment</td>
<td>14</td>
</tr>
</tbody>
</table>
3.9 Cost of not screening

4. Discussion

4.1 Coverage
4.2 Recall rate
4.3 Recall success
4.4 Age of review and age at start of therapy
4.5 Incidence
4.6 Gender ratio
4.7 Diagnoses
4.8 Treatment and follow up
4.9 Outcome
4.10 Comparison of cost of screening to the state versus cost of not screening to the state
4.11 Viability
4.12 The missed cases: Where are they?

5. Conclusion

6. Recommendations

6.1 Education

6.1.1 Delivery unit staff
6.1.2 Mothers
6.1.3 General population

6.2 Facilitated coverage
6.3 Facilitated recall
6.4 Administration
6.5 Cut-off values
6.6 Laboratory protocol
6.7 Neurodevelopmental assessment
6.8 Audit

7. Figures
8. Tables page 43
9. Annexures page 49
10. References page 56
11. Acknowledgements page 61
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital hypothyroidism</td>
<td>CH</td>
</tr>
<tr>
<td>Red Cross Children’s Hospital</td>
<td>RCCH</td>
</tr>
<tr>
<td>Child Health Institute</td>
<td>CHI</td>
</tr>
<tr>
<td>Peninsula Maternal and Neonatal Services</td>
<td>PMNS</td>
</tr>
<tr>
<td>Thyroid Stimulating Hormone</td>
<td>TSH</td>
</tr>
<tr>
<td>Total Thyroxine</td>
<td>tT4</td>
</tr>
<tr>
<td>Free Thyroxine</td>
<td>fT4</td>
</tr>
<tr>
<td>Midwife Obstetric Unit</td>
<td>MOU</td>
</tr>
</tbody>
</table>
1. INTRODUCTION

Congenital hypothyroidism (CH) is one of the most common causes of preventable mental retardation. The frequency of this disorder varies between population groups, but is widely quoted as 1: 3 000 – 1: 4 000 live births. Given this frequency and the need for early treatment to prevent the devastating neurodevelopmental sequelae, newborn screening programmes have been developed and instituted by most industrialized countries. The developing world lags behind, with considerations of poverty, malnutrition and HIV/AIDS demanding the majority of the scarce available resources.

In South Africa, regarded as a developing country, there is no state sponsored thyroid screening programme. However, in 1982, the Peninsula Maternal and Neonatal Services (PMNS) embarked upon a pilot screening programme based on the cord blood of babies born at three maternity units. An audit of the pilot study revealed it to be cost-effective and feasible in the given structure of the PMNS. Subsequently the hypothyroidism screening programme was extended to the entire PMNS.

Two workshops on screening for congenital hypothyroidism held in South Africa, in 1987 and 1992, discussed, inter alia, the outcomes of that programme at those times. At the latter workshop the comment was made:

In developing screening programmes for the detection of congenital hypothyroidism in South Africa several points must be borne in mind. Firstly, most babies are discharged from obstetric units on the day of delivery- later heel-prick blood sampling is not feasible. Secondly, and most importantly, facilities for the tracing and follow-up of positive cases must be established. Thirdly, data from the USA suggest that the initiation of thyroxine therapy per se does not necessarily lead to optimal mental development in the hypothyroid infant: maternal education and frequent biochemical follow-up are a necessary part of the ‘screening package’.

The PMNS programme has run with no further audit until this time. This thesis aims to assess how well these ideals have been met, by looking at the coverage achieved, the recall numbers, the percentage of follow ups achieved, the number and type of
positive hypothyroid cases diagnosed, and the cost and ongoing feasibility of the programme. In addition, by doing this audit, it aims to clarify and highlight any problems within the programme.

For a range of disorders neonatal screening is recommended provided that:

1. There is considered to be a direct benefit to the neonate from early diagnosis;
2. The benefit is reasonably balanced against financial and other costs;
3. There is a reliable test suitable for neonatal screening;
4. There is a satisfactory system in operation to deal with diagnostic testing, counselling, treatment and follow up of patients identified by the test.  

Internationally these criteria are met for congenital hypothyroidism screening; this audit will endeavour to answer whether this programme is viable in its current setting, and whether it should be recommended for the rest of the South African population.

1.1 Congenital hypothyroidism and the history of screening neonates

Primary congenital hypothyroidism arises by either an abnormality in the development of the thyroid gland (aplasia, hypoplasia or ectopia, Figures 1 and 2, pages 38 and 39), or an inability to manufacture and secrete thyroid hormone (dyshormonogenesis). Less commonly, the altered neonatal thyroid function is transient, attributable to the transplacental passage of maternal medication, maternal blocking antibodies, or iodine deficiency or excess. Much rarer secondary hypothyroidism occurs when the pituitary gland dysfunctions, and tertiary when the pathology is at the level of the hypothalamus, Figure3, page 40.

The clinical picture of hypothyroidism is subtle and non-specific. Findings of prolonged neonatal jaundice, macroglossia, delayed closure of the fontanelles, umbilical hernia, constipation, hypotonia and developmental delay may alert a clinician to the diagnosis. At birth and in early infancy the condition may be
asymptomatic. Often the diagnosis is suspected only after the age of 3 months, sometimes as late as school-going age. Because neurodevelopment is dependent on thyroid hormone, the neurological differentiation is already negatively affected well before any clinical changes occur. This impairment in brain development may lead to mental retardation if the hypothyroidism is not treated. However, it has been shown that by early diagnosis and treatment with thyroxine, prior to the onset of signs and symptoms, the mental retardation can be prevented.\(^7,8\) It is on this basis that the neonatal screening programmes have been advocated.

The first screening programme for congenital hypothyroidism was commenced in Quebec in 1974,\(^9\) and now all the states of the USA,\(^10,11\) the majority of European countries and many Asian countries utilise such a programme.\(^9\) The screening programmes in the developed world are usually screens for multiple congenital metabolic disorders, and therefore use whole blood collected on filter paper 2–5 days after birth (this is necessary to pick up the increase in pathological metabolites after birth, once feeding has commenced, in many of the inborn metabolic disorders). In resource poor developing countries, this is neither practical nor financially viable. Mothers and their newborns are often discharged within 24 hours, and it would be expensive and difficult to expect the babies to be returned for the blood taking within a week. The multiple metabolic screen is expensive, and with the extremely low individual incidences, and often limited success of therapy if available, this is not currently indicated in developing countries. Thus, cord blood is the most practical and cost-effective mechanism for screening for hypothyroidism in poorer countries.\(^12,13\)

Cord blood Thyrotropin Stimulatory Hormone (TSH) as the primary screen, with secondary total Thyroxine (tT4) levels has been shown to have acceptable sensitivities and specificities for congenital hypothyroidism screening. However, there are acknowledged higher false positivities than with day 2-5 bloodspots, due to physiological TSH surges at birth.\(^1,14,15,16,17,18,19,20\)
1.2 Materials and methods of screening in the PMNS

1 ml of mixed cord blood is taken at the time of the umbilical cord being cut at delivery. It is labelled with the mother’s name and folder number, the date of the delivery, the baby’s birth weight, and the unit where the delivery occurred, *Annexure 1, page 49*. It is placed in the refrigerator (at 4°C) until collected by the driver, and transported to the laboratory at the Red Cross Children’s Hospital Institute of Child Health (RCCH ICH). This occurs on weekdays, so any specimen collected from Friday evening will wait in the fridge until Monday morning. This process was in place prior to the screening programme, as cord blood was taken for other tests, and the driver/transport does interunit administrative and porter duties. This thyroid screening programme was tagged onto this process to make as little change as possible, both from a logistic as well as economic point of view.

Once the laboratory technologists receive the blood, it is spun down, the plasma pipetted into tubes and stored in the refrigerator. TSH levels are tested Monday to Thursday. If the primary screen TSH is >20mIU/l, a secondary screen tT4 is performed for that sample, on the Friday of that week. *Figure 4, page 41*.

The TSH is tested with the TSH MAIACLONE immunoradioactive assay from BioChem ImmunoSystems, and the tT4 with the Coat-a-Count radioimmunoassay from Diagnostic Products Corporation. Performance details of these assays are provided in *Annexure 2, page 50*.

Any sample with a TSH > 40mIU/l or <1mIU/l is considered urgent and is phoned to the delivering unit, as is any low tT4. Copies of the worksheets recording all the sample details and results are sent back to the delivering unit and the endocrine unit. The delivery unit then reviews the results sheet for any abnormal screens. The TSH is normal if 1-19mIU/l, abnormal if <1mIU/l or >40 mIU/l, and also abnormal if TSH 20-40mIU/l with tT4 < 100pmol/l, *Figure 5, page 42*. 
The delivering unit personnel phone the endocrine unit with the contact details of that abnormal screen. The endocrine sister or volunteer then contacts the mother by telephone if a telephone number is supplied, or by a pro forma letter, Annexure 3, page 54.

The mother or caregiver attends either the Retreat or Mitchell's Plain Midwife Obstetric Unit (MOU), or the Red Cross Children's Hospital endocrine clinic, where the baby is examined, and repeat blood TSH and free Thyroxine (fT4) are sent to the National Health Laboratory Service (NHLS). If these are abnormal the baby attends the RCCH endocrine clinic, where a ¹²³I Tc scan is performed (if it can be done the same day), and thyroxine therapy commenced. Clinical, biochemical and imaging follow up is at the discretion of the endocrinologist, but the usual plan is to confirm the diagnosis at 2 years of age, if not already apparent, by pausing therapy and retesting TSH and fT4 with scintography when indicated.
2. STUDY DESIGN AND RESEARCH METHOD

All births in the PMNS from 01/01/2000 until 31/12/2004 were included in the audit. Data were captured to document all babies recalled for repeat thyroid functions and clinical review following an abnormal cord TSH screen.

The details recorded were:
Date of birth, birth weight, sex, screen TSH and tT4, repeated TSH and fT4, reviewing clinic, follow up TSH and tT4, imaging results, age at initial review, and, if hypothyroid, age at commencement of therapy. Recorded neurodevelopmental progress and/or formal neurodevelopmental assessments of the hypothyroid babies were documented. All data were reviewed and completed as at 31/01/06.

Ethnicity was considered as a desirable datum, but because racial classification in medical records no longer occurs, this was not available.

To assist with data capture and processing, identifying names and folder numbers were also recorded, but to ensure patient confidentiality these were removed once the analysis was completed.

Dr D. Greenfield supplied birth numbers from each of the units within the PMNS.

Prof M. Mann and the laboratory at the RCCH ICH provided TSH screen numbers, the number of fT4 tests done, and the laboratory costs.

Ethics approval was granted by the Research Ethics Committee, University of Cape Town, Annexure 4, page 55.
3. RESULTS

3.1 Coverage

In the audit period 01/01/2000 – 31/12/2004, 140 507 babies were born. 130 389 primary TSH screens were done, which gives a total coverage over the 5 year interval of 92.8%. The coverage has been calculated per unit of delivery, and per year, and described in Table 1, page 43.

3.2 Secondary screening

As per Table 2, page 44, the proportion of tT4 secondary screens to primary TSH screens has remained constantly around the 10% mark (range 8.9 to 11.1%).

3.3 Screen results

Of the 130 389 screens, there were 2 207 abnormal results, as defined under ‘Materials and Methods of Screening in the PMNS’ on page 4.

This gives the percentage of abnormal screens/total screens of 1.69%, which is the recall rate.

Number of hypothyroid cases (*Table 3, page 45*) = 13

Hypothyroid Percentage of total screened = 0.01%

Incidence = 1: 10 000

Hypothyroid Percentage of abnormal screens = 0.59%

= 1: 169
686 patients attended recall, and 65 were reviewed pre-recall, which means only 751 out of 2,207 abnormal screens were reviewed = 34.0%. (Pre-recall reviews occurred when a neonate with an abnormal cord blood thyroid screen was clinically and biochemically reviewed by an attending physician, prior to a recall letter having been sent, e.g. in the case of a premature baby still in a nursery. Such patients may therefore have not attended the Retreat or Mitchell’s MOU, or the Red Cross Children’s Hospital endocrine clinic, as outlined in 1.2 Materials and methods of screening in the PMNS, page 4).

Hypothyroid Percentage of reviewed abnormal screens = 1.73%  
= 1: 58

Since 13 cases were diagnosed from only the reviewed 34% of the abnormal screens, it is possible, if the same incidence occurs throughout the abnormal screens, that there could be 38 cases of hypothyroidism from the total screened group.

Revised for this scenario:

Hypothyroid Percentage of total screened = 0.029%  
Incidence = 1: 3,448

Another 14 of the 751 reviewed abnormal screens had insufficient follow up to exclude hypothyroidism. Therefore, this incidence could be even higher, as these revised figures are calculated on 13 hypothyroid cases diagnosed from the reviewed 751, on the assumption the other 738 cases were reviewed as normal.

Three late diagnosed cases followed up at the RCCH endocrine clinic were born outside the PMNS, Table 4, page 46. As yet, the clinic is not looking after any missed CH from the audit period.
3.4 Gender ratio

All of the cases of congenital hypothyroidism caused by known developmental abnormalities (6 of the screened cases of hypothyroidism, and all 3 of the late diagnosed cases) were female individuals.

3.5 Diagnoses

Of the 13 screened cases of CH, 6 were developmental abnormalities of the thyroid (46%) (5 ectopia +/- hypoplasia and 1 aplasia), 3 were dyshormonogenesis (23%) (of which 1 is a probable uptake defect), 1 was as a result of maternal Graves treated with carbimazole (presumably on an autoantibody basis, since therapy was needed for 6 months, long after the drug effect should have worn off) (7.7%), and in 3 the cause was undiagnosed because they were lost to follow up.

3.6 Treatment and follow up

2 of the screened cases of hypothyroidism commenced therapy within the first month. Table 5, page 47, shows the age in days of first attendance after recall. Three cases did not return for follow up (so no diagnosis was made, and no treatment was instituted), and the treated case 4 was lost to follow up after his 4th visit. (Cases marked with asterixes in Table 3, page 45).

3.7 Neurodevelopmental outcome

In Table 6, page 48, the developmental assessment of each of the CH cases has been detailed.
3.8 Cost of the screening programme

The cost was calculated as for the last year of the audit. An attempt was made to include all the costs related to the screening programme, from the time of specimen collection, to the time of review. When possible, these have been defined in monetary terms, but when not possible, they have been descriptive.

3.8.1 Consumables:

The consumables are for the most part the laboratory testing kits. The cost incurred is calculated as follows:

Number of TSH screens done in 2004 = 28 173
Number of tT4 secondary screens done in 2004 = 2 794
R9.21/test/baby for TSH (as per Prof M. Mann, RCCH Laboratory, ongoing personal correspondence)
R16.82/test/baby for tT4 (as per Prof M. Mann, RCCH Laboratory, ongoing personal correspondence)

Cost of TSH kits = 28 173 x R9.21 = R259 473.33
Cost of tT4 kits = 2 794 x R16.82 = R46 995.08
Total kits cost for 2004 = R259 473.33 + R46 995.08 = R306 468.41
Cost of consumables per baby screened (averaged to include TSH and T4)
= R306 468.41 / 28173 = R10.88

Other consumables including stationery and stamps have not been calculated.

3.8.2 Staff:

The maternity staff who collect the cord samples have not been included in the costing. They already collect cord samples as part of their routine care. The additional time taken to fill in the paperwork and send the samples is difficult to cost, but would account for approximately 2 minutes per baby of the midwives' time.
The situation during the audit period was that two laboratory technologists conducted the thyroid screen work, along with other duties (including in vivo tests such as glomerular filtration rates, preparing radiopharmaceuticals such as sucralfate, helping with radiation safety, etc.). It has been calculated that the work could be done by one technologist, if leave and other absence could be covered by other laboratory staff. The cost of this technologist to the state as at 2004 was R105 018 per annum (salary level 8).

The staff who process the recalls include the endocrine nursing sisters and volunteers at RCCH, and the paediatric doctors at the delivery units. The endocrine staff, at the time of the audit, were committing 2 hours per week to sending recall letters and making recall telephone calls. The scrutiny of returns and attempts to follow up on defaulters or non-replies was neglected due to lack of time, and commitment to other clinical responsibilities. To cost the necessary time to do the recall job correctly, it is estimated that a full time junior clerk would be competent in this regard. This would cost the state the equivalent of a level 4 salary, which was R46 353 per annum as at 2004.

The staff at the delivery units spend approximately 15 to 30 minutes per week checking results, identifying contact details and phoning or faxing them through to the endocrine team. This time is considerably longer when the contact details need to be sourced from the delivery units’ records before they can be passed on to the endocrine team (personal correspondence Dr M.Kroon, MMH, February 2007).

3.8.3 Equipment:

A centrifuge and gammacounter are required equipment in the laboratory. Both these items last approximately 20 years, and can be used by the laboratory for other specimens.
3.8.4 Additional tests:

751 out of 2,207 abnormal screens were actually reviewed.

The reviewed cases all have a TSH and fT4 done by the NHLS. The costs of the tests were those charged by the NHLS to the government in 2004. For this calculation, it is assumed that each reviewed case had a review TSH and fT4 requested. (Although, in practice, this was not the case. Some reviewed individuals were so old at the time of review, that the clinician decided on clinical grounds that biochemistry was not necessary. For costing purposes, this situation is negligible, and in a screening programme running efficiently, should not occur).

R117.45/TSH test (NHLS, 2004)
R98.94/fT4 test (NHLS, 2004)

TSH = 751 x R117.45 = R88 204.95
fT4 = 751 x R98.94 = R74 303.94

Total = R162 508.89

Had all the abnormal screened cases attended review, the costs would have been:

TSH = 2,207 x R117.45 = R259 212.15
fT4 = 2,207 x R98.94 = R218 360.58

Total = R477 572.73

This is the end point of the screening programme as far as tests go. The investigations that occur thereafter, including ultrasound and scintography are not unique to the screening. They are rather part of the management of the patient, and would occur whether the diagnosis was made from screening or from clinical suspicion. Costs of these investigations and follow up thyroid tests have therefore not been included.
Cost of screening for 2004 = R306 468.41 consumables
R105 018.00 technologist
R 46 353.00 clerk
+ R162 508.89 review tests
R620 348.30

Cost of screening for 2004 had all abnormal screened cases attended review

= R306 468.41 consumables
R105 018.00 technologist
R 46,353.00 clerk
+ R477 572.73 review tests
R935 412.14

If one uses the incidence of 1: 10 000, which was the actual rate calculated in the audit period, then there would have been 2.8 cases identified in 2004, leaving a cost of screening and detection of R221 552.96 per case. If the incidence of 1:3 445 is used, which is the incidence corrected for the non-attendance of abnormal screened cases, then there should have been 8.2 cases identified. If all the abnormal screen cases had attended and these 8.2 cases were detected, the cost of screening and detecting would then have dropped to R114 074.65 per case.

Per baby, the screen cost R22.02 in 2004. This would have increased to R33.20 had all the recalled babies attended review and had review TSH and fT4 tests.
3.8.5 Treatment:

The cost of treating a child with hypothyroidism once diagnosed early is very little. Eltroxin (thyroxine replacement) is inexpensive, even at adult replacement amounts.

At a dose of 25 microg/day Eltroxin (the average dose for the first year of therapy), it would currently cost the state R3.17 per month to treat a baby with hypothyroidism (personal correspondence, RCCH Pharmacy, February 2007).

3.9 Cost of not screening

The cost of not screening is extremely difficult to narrow down to a single rand figure. Many factors need to be taken into account, so for the most part, this will be a descriptive exercise.

It is known that an untreated case of hypothyroidism is likely to develop severe neurodevelopmental impairment (the old term for such a person is ‘cretin’), but the degree to which an individual is disabled when diagnosed and treated as a child is widely variable, from only mildly to fully impaired. This depends largely on the diagnosis and severity of the hypothyroidism. Aplasia fares the worst, since levels of thyroxine in affected individuals are extremely low, even since in utero. When accounting for costs to the state, the degree to which an individual is affected is important, as resources that must be made available to the individual vary according to needs.

Therefore costing for not screening needs to examine expenses incurred with respect to:

1. home care for a severely impaired individual
2. home care plus special schooling for the individual moderately impaired
3. special classes within mainstream schools for individuals mildly impaired
4. care dependency / disability grants
5. loss of income for the individual and/ caregiver – this incurs costs to the state as there is loss of tax revenue, plus the lack of income leads to requirement of access to other state services, e.g. subsidised housing and medical care

6. occupational therapy and other remedial therapy

This exercise in accounting for the cost of the lifetime of an individual with severe mental impairment has not been undertaken as of yet. Personal communications with the Children’s Institute, University of Cape Town (CI UCT) (Lizette Berry, Child Poverty Programme, February 2007), indicate work done on investigating access to grants, and the *Means to Live* as outlined in the Child Gauge. 22,23 There has been no focus on the cost of special needs, either to the state or to the individual. The Children’s Bill makes provision for the costs of disabled and chronically ill children in children’s homes, and addresses the care dependency and foster grants, but does not address special needs as a whole entity. It focuses on a child’s needs in its entirety, and considers the disabled child as having the same needs to be met as an unaffected child (e.g. food and shelter) (personal communication, Sue Philpott, Disability task team for the Children’s Bill process, February 2007). It also does not account for the fact that the disabled child may need more focused, long term care, and may not graduate out of the need as does a normal child. Personal communication with the Neurodevelopment Unit at RCCH (Dr V. Ramanjam, January 2007) also established that there has not been any local costing exercise as described above.

In ‘Prevention of Congenital Hypothyroidism: Economic Implications’, 4 the authors used a combination of options, from 1. care at home, 2. care and rehabilitation centre, 3. special school education, 4. hostel fees and 5. after-care centre. They used a life expectancy of 40 years and managed to calculate, using variable combinations of the above, an amount of R330 000 the lifetime cost of looking after a mentally impaired person. Given that these calculations were from 1987, this amount would be much inflated in 2004. Unfortunately the authors did not reference their sources, so the 2004 equivalents could not be costed. Also, of note, is that South Africa has undergone political change since 1987. Delivery of health is no longer divided by
racial lines, and there is a greater emphasis on primary care. Quoted costs as at 1987 would be very different in 2004, not only due to inflation, but also due to the different range of facilities available and a different approach to looking after the mentally impaired.

One cannot put a value on the ethics involved in screening for a preventable cause of mental retardation. However, given that the South African society is becoming more litigious, it will be a matter of time before the courts may be forced to decide on a monetary sum as a settlement in a liability case against the state. While not entirely the same situations, two recent instances mentioned in the press have highlighted this fact. In Van Antwerp versus Hans Graser, André Swart and Jacobus Scholtz, the claimant received R99.6 million for the medical negligence of the doctors resulting in a child’s brain damage, \(^{24}\) and in a suit against Michael Wright, and Medi-Clinic Limited, the court has settled for a couple who have a brain damaged child as a result of negligence. The sum of the award in this case was not published, but the claim is known to be for R11 million. \(^{25}\)
4. DISCUSSION

4.1 Coverage

From Table 1, page 43, it is clear that through the 5 year audit period, the delivery units tend to have similar coverages from year to year, although Gugulethu MOU, with the lowest coverage generally, has shown the greatest improvement in coverage. The second lowest coverage overall is at Khayelitsha MOU, which shows no real improvement in coverage from 2000 to 2003. Note that for Mowbray Maternity Hospital (MMH) in 2003, and again 2004, along with Vanguard Drive, Khayelitsha, Hanover Park, Mitchell’s Plain and Retreat MOUs, the screened numbers exceed the reported birth numbers.

This is of some concern, as either the delivery units have underreported their numbers, or the laboratory has reported higher screened numbers than actually done. The computerised database for the PMNS was being upgraded during the latter half of the audit period, so this could account for inaccuracies in the birth numbers (personal correspondence Dr M. Kroon, MMH, February 2007). There is no motivation for over-reporting by the laboratory, and random review of various samples of tests on random days shows no duplication or exaggeration of numbers.
This irregularity between possible number of births (reflected by the number of tests) and number of reported births by delivery units has economic and statistical implications. Resources allocated to departments depend on the numbers of patients attended to and the number of deliveries, so by under-reporting birth numbers, the delivery unit may receive less funding in future financial years than justly deserved. Accurate birth numbers are needed for many statistical analyses including neonatal and infant mortality rates, assessing the state of the Health Services, the requirements of the Social Services and for projecting the population profile for future resource allocation by the state. In order to establish the incidence and prevalence of communicable and preventable diseases, accurate population numbers are required, and these commence with birth reports.

There is considerable variation in coverage between the delivery units. Some units have consistent coverage >90%, while others are very low, with no increase in uptake over time.

The midwives and delivery personnel are integral to successful coverage of the whole population. There has been no direct comparison of levels of staff qualifications and ongoing education between the units, but it is interesting to note that some of the primary facilities (Hanover Park and Retreat MOUs) have better coverage than a secondary level institution (New Somerset Hospital) (Table 1, page 43). Gugulethu and Khayelitsha, both areas of previously disadvantage populations, have the lowest average coverage, but Gugulethu has shown the most improvement in coverage over the audit period.

Ongoing education of the delivery staff, with visible reminders in the delivery units could improve coverage. Standard forms inserted into every antenatal folder, with the mother's sticker/details already supplied pre-delivery would assist with the workload at the time of delivery.
Established standard of care is that 100% of the eligible population is screened. It is therefore important to conduct an audit intermittently to ascertain whether this has been achieved.

It is also important that every delivery has a noted screen. At the time of the audit there was no cross-referencing of deliveries to screens. All documentation was on paper, which made access to birth and screen numbers difficult. It was difficult to find an individual screen result after the fact. Once the child has a name and different folder number allocated, there is no link between the child's result and the child, as the result is under the mother's name and reference number.

Computerised databases could assist in this regard. The number of deliveries in a unit per week can be cross-referenced by the number of screens done for that unit for that week. If there is a shortfall, the results can immediately be compared to identify the non-screened individuals.

As a matter of record, the delivery units should have access to all the screen results, which should be filed into the patients' folders. All babies should be reviewed at 6 weeks at the time of their first immunization, and this opportunity could be used to ensure a screen result was filed for that baby.
4.2 Recall rate

The recall rate of 1.69% is high for a screening programme, although similar to other published programmes.\textsuperscript{15,17,26} It would improve cost effectiveness to decrease this to the generally accepted 0.3%.\textsuperscript{18} To do this, cut-off values will need to be changed. Current cut-off levels have been ‘safe’ in that it is considered no false negatives will occur. However, these levels concede a high false positive rate.\textsuperscript{27} Fifty-eight reviews have to occur to detect one hypothyroid case. If the upper limit cut-off for TSH is changed to $<1$ and $\geq 50$ mIU/l for immediate recall, with TSH $20-49$ mIU/l and tT4 $<100$ pmol/l for recall, none of the detected hypothyroid cases would have been missed. 129 of the 686 reviewed recall letter cases would have then been classified as normal and not recalled. This would have dropped the recall rate by 18.8%, so that 43 reviews would have detected one hypothyroid case. While this would still give a recall rate of 1.37%, the cut-off levels could again be reviewed after one year to further lower this rate.

4.3 Recall success

The 34% review rate is very poor, and is the greatest problem identified by this audit. In attempting to identify the reason for only 715 of the 2207 abnormal screens being reviewed, the following issues have come to light:

1. There is absolutely no parent education about the screening programme or hypothyroidism.

2. The contact details supplied by the delivery units are often incorrect.
   Sometimes there is an inaccuracy in relaying the information, and on review a correct address is supplied (which causes avoidable delay in recall). More often, the actual address supplied by the mother is incomplete or inaccurate. Anecdotally, the reasons suggested for this are: a) Fear of being billed for the perinatal services; and b) Fear of being turned away from the chosen delivery unit because of living ‘out of the catchment area’ – a false address within the desired area is supplied.
3. The mother is no longer residing at the communicated address. With obstetric care perceived as better in the urban Cape Town area than the rural areas where many of the mothers live, migration to the city for the delivery occurs. Mothers than return to their homes shortly after the delivery, and are no longer contactable.

4. The recall letter is in English. While it is gently worded and explained, even when understood, it often elicits a response of fear and surprise.

To remedy this situation, the biggest contribution would be education of the mother. There should be a policy of educating the mother during an antenatal visit about the screen, with basic information supplied about hypothyroidism and the ability to prevent mental retardation if treated timeously. This information should be available at least in English, Afrikaans and Xhosa in the PMNS.

All mothers should be able to have access to their child’s result. They should be aware that they would only be contacted if the result were abnormal and needed review to determine if the baby did indeed have hypothyroidism. They should also be aware that they should supply contact details where they would be contactable for at least the 6 weeks following the birth of the child. In the South African situation, at least here in the Western Cape, there is a high percentage of cellular phone users compared to landline telephone users. A cell number would be most helpful in that it is usually transportable with the mother, and contact could be made by text messaging if the mother is not available to take the call immediately.

The education of the mother is the greatest way to provide incentive for correct contact details to be supplied, and for attendance on recall and follow up. This point has been highlighted in a publication from Kuala Lumpur, where they too had a low recall success. 20

What is also apparent is that there is a high default rate of those who already attended review. One can only speculate the reasons for this 31% non-attendance at follow up.
Possible concerns are:

1. Language and educational barriers.

The majority of the doctors speak English and Afrikaans and may only have limited Xhosa knowledge. While interpreters are available at RCCH, they are often in demand and hurry from one interview to another. With the large recall rate, clinics can be very busy, so, at least at the first review visit, there is not a lot of time to educate the mother about the screen and the condition. With the rate of only 1 case per every 58 reviews actually having hypothyroidism, this also impacts on the desire to spend time educating the mother when the child may not have the condition. This review visit is unfortunately the mothers’ first contact with the programme, and this should therefore be a time of great input. The default rate may be related to the fact that the mother does not understand the screening programme or the condition, and may well be afraid of the result and the diagnosis.

The antenatal education of the mother would at least prepare her for the recall, and allow for some prior insight into the concept of a screen. It would then be easier to discuss the implications of the review result at the review visit, and allow for more detailed explanations of the need for adherence to follow up plans.

2. Migration.

As mentioned above, mothers move to Cape Town for their obstetric care, and then move back to their rural homes after the delivery. They may attend the review in Cape Town, but may be afraid to mention that they will be in a different province at the time of follow up. If the clinicians are alerted to this fact, they can include this possibility in the discussion with the mother at review, and at least offer the mother the chance to attend her local paediatric clinic for the follow up. This can then be facilitated by providing the mother with a referral letter, supplying all the relevant details for appropriate follow up.
4.4 Age of review and age at start of therapy

As already described in results, neither the age of review (Table 5, page 47), nor the age of start of therapy (Table 3, page 45) falls within accepted standards of care. This identifies the second greatest problem with the current programme.

The interval between screen and review depends on several factors:

1. The transport of screen specimens from delivery unit to laboratory;
2. The laboratory processing time;
3. The communication of the results to the delivery unit personnel;
4. The reviewing of the results and communication of abnormal results and contact details to the endocrine unit staff;
5. The processing of recalls by telephone and letter by the endocrine unit staff;
6. The postal system delivering the recall letters;
7. The mothers receiving and understanding the recall information, and then attending review clinic;
8. The endocrine staff confirming attendances, detecting the non-attending cases, and endeavouring to recall them.

Many of these points were similarly raised in 1988 about a pilot screening programme in Pretoria, South Africa. 29 Currently, anecdotally, steps 1 through 4 are not problematic. The problems begin with incomplete or inaccurate contact details, as the endocrine nursing staff have to allocate a lot of their time to finding the correct information. Because the endocrine nursing staff have other responsibilities, there is often a delay in steps 5 and 8. These could be addressed by allocating the responsibility to one clerk, who is dedicated to attending to these tasks daily. The clerk does not have to have this as their sole responsibility, but must be accountable for the process.
Step 6 is not easily changed, but by mothers providing telephonic contact details, may be avoided.

Step 7 can be improved by parent education at antenatal and postnatal levels, as discussed above.

The age of starting therapy is dependant on the age at first review, and on the clinician reviewing results, investigating the case further and deciding on a treatment schedule. Currently, most of the results are checked the week following review, as the endocrine clinic is held weekly. The laboratory staff may contact the endocrinologist with a markedly abnormal result on the day of the test, but this is as a result of personal initiative, not protocol. The abnormal review result is then conveyed to the mother who is asked to attend the soonest endocrine clinic day, when a scan is performed, if possible, and treatment is commenced. If the results are borderline, the mother is given an endocrine clinic follow up date to attend for further review.

More recently, when markedly abnormal results have been conveyed to the endocrinologist early, an attempt has been made to have the mother and child return the following day, for further investigation and commencement of therapy.

To facilitate this procedure, it should be made policy that the laboratory staff alert the clinician and/or clinic to an abnormal result on the day of the test. The reviewing clinic should have a list of the reviewed cases, and ensure that the results are checked the day after the test, so that the abnormal cases can be contacted immediately for prompt investigation and treatment. The clinicians have many responsibilities and have clinics in different locations, so may not be able to review the results of the individuals they examined in the clinic. Therefore it would serve the programme better to have one person responsible for this. It is suggested that the same clerk who does the initial recalling can be trained to identify abnormal review tests, and process the contacting of such cases immediately.
4.5 Incidence

The incidence of congenital hypothyroidism is high in certain Middle Eastern communities, especially where consanguinity is common, at a rate of between 1: 900 and 1: 1500. 17, 26, 29 As previously mentioned, the general worldwide population incidence is 1: 3 000 – 1: 4 000. It has been previously identified that African, or African-origin, populations have lower incidences, as low as 1: 32 000 (as an aside, the Japanese incidence is also very low at 1: 8 000 ’).

Because South Africa no longer classifies race in medical records, the ethnicity of the population and of the detected hypothyroid cases is not known. Cape Town has a diverse population, with people of many different ethnicities. The widest spread is of Xhosa, European and Malay ancestry, but with additional Indian and other African groups contributing to the racial profile. The incidence (revised for the actual cases detected in the reviewed screened population) is 1: 3 448, which, of interest, falls in the middle of the ‘worldwide’ population incidence.

This may not be the incidence for South Africa as a whole because the ethnic mix does not occur to such a wide extent in the rest of the country, but certainly holds the Cape Town programme important. The South African incidence is not known, so providing ethnicity for an auditable time period to establish ‘African’ incidence would be advisable. This would contribute towards deciding whether such a screening programme would be viable for the rest of South Africa. Of note, the incidence required to make a screening programme practical has been quoted as > 1: 15 000 30, which makes this audit incidence fall well within that range, even when not corrected for the non-reviewed cases.

4.6 Gender ratio

The audit shows all CH cases caused by developmental abnormalities were females. Table 3, page 45. The literature suggests a female: male ratio of 2: 1 in these conditions. 31, 32, 33 The small number of cases (9) from this audit may account for the discrepancy.
4.7 Diagnosis

This audit finds a slightly higher ratio of dyshormonogenesis (23%) when compared with rates of thyroid ectopia (46%), thyroid aplasia (33%), and dyshormonogenesis (11%) in the Australian study; athyreosis (45%), thyroid ectopia (24%) and dyshormonogenesis (17%) in Saudi Arabia; and thyroid dysgenesis (80.9%), which consisted of 40.4% athyreosis, 4.3% hypoplasia, and 36.2% thyroid ectopy, with thyroid dyshormonogenesis accounting for 18.9% in Thailand.

4.8 Treatment and follow up

Only 2 of the screened cases of hypothyroidism commenced therapy within the first month, considered essential standard of care. It must be noted, however, that the decision to start therapy was at the clinician’s discretion. In some cases the initial review results were borderline so therapy was delayed to see the trend to exclude mild transient hypothyroidism.

Table 5, page 47, shows the age in days of first attendance after recall. There is no average within the accepted standard of 20 days.

Three cases did not return for follow up (so no diagnosis was made, and no treatment was instituted), and the treated case 4 was lost to follow up after his 4th visit. (Cases marked with asterixes in Table 3, page 45).
4.9 Outcome

There has been no formal audit of intellectual outcome of this programme’s detected hypothyroid cases. The cases identified were not yet in formal schooling while this audit was conducted, and so school competency had not been assessed. However, anecdotally, it was noted that during the time frame of the audit, only one of the detected cases had been referred for neurodevelopment assessment. In fact, the cases that were born outside of the screening area (*Table 4, page 46*) were more likely (2 out of 3 cases of “outside” versus 1 out of 13 “screened” cases) to have been formally neurodevelopmentally assessed. This is presumably because their presentations included developmental delay, whether as a consequence of their hypothyroidism, or secondary to other pathological process. Case 2, Table 4, was 0.8kg at birth, and Case 3, Table 4, was dysmorphic and also had tuberculous meningitis, so direct comparisons of neurodevelopmental outcomes in this small group is impossible.

While screening programmes, by early detection and treatment of congenital hypothyroidism, have eradicated ‘cretinism’ as a cause of mental retardation, there may still be subtle neurological sequelae to congenital hypothyroidism that need to be monitored. The comments made by the clinicians in the notes of each of the screened hypothyroid cases suggests normal development, but bearing this possibility of subtle sequelae in mind, each individual diagnosed with congenital hypothyroidism should have neurological assessment as part of the routine follow up.

4.10 Comparison of cost of screening to the state versus cost of not screening to the state

In “Prevention of Congenital Hypothyroidism: Economic Implications”, the authors come to the conclusion that (in 1987) it would cost R12 000 to detect and treat a child successfully, compared to the cost of R330 000 were this not done. A cost: benefit ratio of 1:28 was calculated. They therefore advocated the congenital hypothyroid screen as a cost effective programme.
Other countries have also attempted such a costing, noting the same difficulty in costing the 'not screened' situation, but coming to the same conclusion that screening is cost effective. ¹

From the above investigation of current costs in South Africa, while one cannot make a cost: benefit ratio, it is apparent that the screen is indeed cost efficient. The current cost of the screen per hypothyroid case actually identified is well below even the lifetime cost of looking after such an individual not screened in 1987.

4.11 Viability

Returning to the points made in the introduction, for a screening programme to be recommended:

1. There is considered to be a direct benefit to the neonate from early diagnosis;
2. The benefit is reasonably balanced against financial and other costs;
3. There is a reliable test suitable for neonatal screening;
4. There is a satisfactory system in operation to deal with diagnostic testing, counselling, treatment and follow up of patients identified by the test.

From this audit and literature review, it is clear that points 1 and 3 are easily met. The 2nd point has been debated above, and appears to have been defended. The 4th point is the current point of contention.

Considerable work needs to be done to improve the recall success and follow up. The greatest contribution would be by incorporating education of the mothers into the antenatal care. This would directly involve the mothers in the programme, and would establish a background population knowledge and understanding of the screening programme.
4.12 The missed cases: Where are they?

A last comment needs to be made as to why the RCCH endocrine clinic is not looking after any hypothyroid babies missed by the screening programme. Considering only 34% of the abnormal screened cases were reviewed, it would be expected that there were at least another 25 cases of congenital hypothyroidism born during the 5-year audit period. Possible reasons for their conspicuous absence from the endocrine clinic are:

1. They have been diagnosed clinically, but are being treated elsewhere. This is possible, as the migratory nature of this population group has already been alluded to. However, the diagnosis would most likely be made later than the standard of care expects, so this is still not the ideal.

2. They have not yet been diagnosed. This is a strong possibility, since, as already mentioned, the clinical diagnosis of hypothyroidism is difficult, and the clinician has to have a high index of suspicion. The children born in the audit period were not in formal schooling yet, and therefore developmental delay and intellectual impairment may not yet have been discovered.

3. They have died. A small percentage of congenital hypothyroid cases have other congenital abnormalities that may be life threatening, and may have result in their demise prior to the diagnosis of hypothyroidism being made. The HIV/AIDS epidemic may be decreasing the screened population base before the hypothyroidism diagnosis is made. The number of HIV-related deaths from the audited population group is not known.
5. CONCLUSION

In the audit period of 01/01/00 – 31/12/04, 140 507 babies were born. 130 389 primary TSH screens were done on cord blood, which gives a total coverage of 92.8%. 13 cases of congenital hypothyroidism were detected out of 34% of the 2 207 abnormal screen results, resulting in a corrected incidence of congenital hypothyroidism in the PMNS during this audit of 1: 3 448. This falls within published international population incidences.

The cost of the screen was R620 348.30 in 2004, at R22.02 per screened baby, and equals approximately R221 552.96 per hypothyroid case detected. This is economically viable, as the described costs of a case of congenital hypothyroidism not being detected is much more, regardless of the ethical consideration of not detecting a preventable cause of intellectual impairment.

Problems identified by the audit include:

1. A very poor 34% recall success, and a high 31% default rate.
2. No education made available to mothers or the general public about the screen and congenital hypothyroidism.
3. An unacceptably long delay before review, and starting of treatment beyond the acceptable first month.
4. No standard of practice for neurodevelopmental follow up.

Despite these problems, other positive factors were elicited. Some of the delivery units have extremely good coverage; and the process of taking the cord blood sample, transporting it to the laboratory and processing it, is functioning well.
Formalising the data capturing of samples and results would improve the ability to trace abnormal screened cases for recall. Allocation of responsibility to a specific clerk for recalling both the abnormal screened cases as well as the abnormal reviewed cases would ensure continuity and dedication to this task.

Education of the mothers, and general population awareness of the screening programme, would improve recall success and follow up rates.

Finally, an audit should be undertaken after regular intervals to ensure the functioning of the screening programme and the appropriate usage of resources.
6. RECOMMENDATIONS

Out of the discussion come several recommendations.

6.1. Education

6.1.1. Delivery unit staff

By educating the staff responsible for the screen samples, one would improve coverage of the programme. Ongoing education, as well as feedback of coverage figures to the delivery unit staff is vital. Reminders such as posters in the delivery rooms and stickers on charts would also further increase uptake by the staff.

6.1.2 Mothers

In this audit, as well as referenced publications, it is apparent that the screening programme success depends on the ability of the system to recall and follow up cases. Without a doubt, the most important factor in the successful implementation of the screen is the mothers’ education. The contact details, the response to a recall letter or telephone call, the response to review results, the attendance at follow up, and the treatment of the child all depend on the mothers’ knowledge, understanding and acceptance of the screening programme and its intended outcome. Therefore, the education of mothers at antenatal clinics and at the time of sample collection (in this case, at delivery) must be made protocol. A policy needs to be drafted to ensure that every mother who attends antenatal clinics has received both verbal and written information on CH and the screening programme. This information should be available at least in English, Afrikaans and Xhosa in the PMNS. The screen should be mentioned in the one-on-one patient-carer consultation, but should also be promoted in the group education opportunities that arise in the waiting rooms.
The International Society for Neonatal Screening (ISNS) has made a statement, which emphasises these points:

1. Provision of information on screening is a responsibility shared by the screening program and health care professionals (midwives/pediatrician [sic]/nurse);

2. Provision of information about the screening program to professionals in charge of screening is essential so that they can pass it on to families;

3. The parents must be guided through the whole screening process. For each step, verbal as well as printed information (leaflets) must be given by the professional. This information should be such as not to alarm the parents. It should include screening objectives from a health point of view, diseases screened for, sampling procedure, consent process (when applicable) and notification of results;

4. The optimal timing to provide the first information is during the prenatal period and should be repeated at time of sampling. When applicable, give the families enough time before actual consent is requested;

5. Leaflets (or other material such as video/CD/DVD) should be available in different languages as to accommodate various ethnic backgrounds of parents.  

6.1.3. General population

With time, as more mothers become educated through antenatal clinics, a background level of knowledge about the screening programme will become evident. This will need to be enhanced by direct education, e.g. through newspaper articles. By having the general population educated about the programme, it makes it easier for the mothers to attend recall and follow up, as well as allows for immediate family and friends to understand and support the process. By having increased general population knowledge, there is also a greater chance of cases missed by screening being discovered and tested, as there is more chance of a carer or family member enquiring after a child’s results.
6.2 Facilitated coverage

To assist midwives and delivery staff at the time of delivery, an admitting clerk can insert the required screening form already labelled with the mothers’ details into the antenatal folders. This would serve as a reminder to take the screen sample, as well as cut down on paperwork at the delivery staff’s busiest time.

6.3 Facilitated recall

By educating the mothers, one will encourage accurate contact details to be recorded. A permanent physical address must be documented. To further facilitate rapid recall, it is recommended that cellular telephone details are also recorded, as text or voice messages can be used for recall, in addition to posted letters. Again, education of the mother at the antenatal clinic would prepare her for such a message, and with the knowledge and understanding of the screening process, would allow for less stressful recall.

It is recommended that the mothers be given the opportunity to disclose that they may move out of the area the PMNS serves after the delivery, so that they can still be contacted if recall is necessary, and an appropriate referral can be made.

At the 6 week postnatal check up that all babies are supposed to attend, it is recommended that the healthcare worker note the screen result. Missed screens can then be identified, and abnormal screens that have not yet attended review can be immediately sent for review. This would only be feasible if there is a computerised database from which one can access the screen results.
6.4 Administration

There must be an individual assigned responsibility for the recall of abnormal screened cases, the cross-referencing of screens to births and the recall of abnormal reviewed cases. It is recommended that a junior clerk fulfil this requirement. In addition, a computerised database is required, to make cross-referencing possible, to facilitate recalls, as well as to allow for easy access to screen results.

Once a computerised database is available, it can be linked to the delivery unit database, so that the clerk can access the contact details of the mother. By doing this, one will cut out the need for the delivery unit paediatric staff to read the screen results, find the contact details of the mother and communicated them to the endocrine unit. This will then shorten the time taken for recall, as well as free the delivery unit paediatric staff for other duties. Importantly, the computerised database must be able to record whether the screened baby is still in the delivery unit, whether the baby is dead, or whether it has been discharged, so the clerk can make the appropriate notification.

6.5 Cut-off values

It is recommended that the cut-off limits be changed. The new limits should be for TSH <1 and ≥50mIU/l for immediate recall, with TSH 20-49mIU/l and tT4 <100pmol/l for recall. After a year, a repeat audit can again advise whether it is safe to further lower the recall rate.
6.6 Laboratory protocol

It must be made standard protocol that all abnormal review TSH and fT4 results are communicated to a dedicated person (the recommended clerk in 5.4) verbally immediately the result becomes available. The laboratory staff can be notified that the sample is a review TSH and fT4 by the doctors requesting the test. Written confirmation of the result must then follow. This will enable rapid recall of hypothyroid cases, allowing for prompt investigation and commencement of therapy.

The clerk responsible for the screen recalls and reviews should have a list of the reviewed cases from that week, and should ensure that all the reviewed cases results are checked the day after the test. In this way, any abnormal results not conveyed by the laboratory will be identified and can be instantly acted upon.

6.7 Neurodevelopmental assessment

It must be made protocol that every child identified with CH must be referred for neurodevelopmental assessment and follow up. The neurodevelopmental team must be made aware of the current research into ongoing neurological sequelae of CH.

6.8 Audit

Additional data to be examined at future audits should be:

1. Time taken for transport of the screen samples to the laboratory;
2. Time taken for the primary screen test at the laboratory;
3. Time taken for the secondary screen test at the laboratory;
4. Time taken for the laboratory to notify delivery unit staff/clerk of abnormal screen results (depending on whether above changes in 5.4 have been implemented);
5. Time taken for delivery unit staff to inform endocrine staff of abnormal results and contact details (if above changes in 5.4 have not been implemented);
6. Time taken for endocrine staff/clerk to recall telephonically or by mail the abnormal screen individuals;

7. Time for thyroid function to normalise after commencing therapy;

8. Neurodevelopmental outcomes.
FIGURE 1 Development of the thyroid gland.
A-C, Schematic sagittal sections of the head and neck regions of 4-week, 5-week, and 6-week embryos, illustrating successive stages in the development of the thyroid gland. D, Similar section of an adult head and neck, showing the path taken by the thyroid gland during its embryonic descent (indicated by the former tract of the thyroglossal duct).
(From The Pharyngeal Apparatus p166, Before We Are Born: Essentials of Embryology and Birth Defects, 6th Ed, Moore Persaud)
FIGURE 2 Sketch of the head and neck showing the usual sites of ectopic thyroid tissue. The *broken line* indicates the path followed by the thyroid gland during its descent, as well as the former tract of the thyroglossal duct.

(From The Pharyngeal Apparatus p169, Before We Are Born: Essentials of Embryology and Birth Defects, 6th Ed, Moore Persaud)
FIGURE 3 Hypothalamic-Pituitary-Thyroid axis.
TRH-Thyrotropin Releasing Hormone, TSH-Thyroid Stimulating Hormone, 
T4-Thyroxine

- produces
- stimulates
Primary Screen: TSH

1mIU/l - 19mIU/l

Normal
No further action

<1mIU/l
≥20mIU/l

Abnormal
Perform Secondary Screen: tT4

TSH-Thyroid Stimulating Hormone, tT4-total Thyroxine

FIGURE 4 Algorithm for Laboratory Screening.
TSH-Thyroid Stimulating Hormone, tT4-total Thyroxine

FIGURE 5 Algorithm for Cord TSH results and recall.
8. TABLES

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<td>6471</td>
<td>4874</td>
<td>1724</td>
<td>2287</td>
<td>1826</td>
<td>2432</td>
<td>1600</td>
<td>2396</td>
<td>28185</td>
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<td>2002s</td>
<td>3840</td>
<td>6461</td>
<td>4467</td>
<td>1591</td>
<td>1737</td>
<td>1730</td>
<td>2221</td>
<td>1484</td>
<td>1864</td>
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<td>83.9</td>
<td>99.8</td>
<td>91.6</td>
<td>92.3</td>
<td>76.0</td>
<td>94.7</td>
<td>91.3</td>
<td>92.8</td>
<td>77.8</td>
<td>90.1</td>
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<td>2003b</td>
<td>4524</td>
<td>6610</td>
<td>5295</td>
<td>1780</td>
<td>1983</td>
<td>1913</td>
<td>2530</td>
<td>1697</td>
<td>2395</td>
<td>28727</td>
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<tr>
<td>2003s</td>
<td>4140</td>
<td>7097</td>
<td>4873</td>
<td>1627</td>
<td>1532</td>
<td>1742</td>
<td>2271</td>
<td>1616</td>
<td>1962</td>
<td>26860</td>
</tr>
<tr>
<td>%</td>
<td>91.5</td>
<td>107.4</td>
<td>92.0</td>
<td>91.4</td>
<td>77.3</td>
<td>91.1</td>
<td>89.8</td>
<td>95.2</td>
<td>81.9</td>
<td>93.5</td>
</tr>
<tr>
<td>2004b</td>
<td>4577</td>
<td>7323</td>
<td>5414</td>
<td>1118</td>
<td>1392</td>
<td>1122</td>
<td>1567</td>
<td>1105</td>
<td>2342</td>
<td>25960</td>
</tr>
<tr>
<td>2004s</td>
<td>4090</td>
<td>7582</td>
<td>4605</td>
<td>1459</td>
<td>1766</td>
<td>2123</td>
<td>2559</td>
<td>1635</td>
<td>2071</td>
<td>27890</td>
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<tr>
<td>%</td>
<td>89.4</td>
<td>103.5</td>
<td>85.1</td>
<td>130.5</td>
<td>126.9</td>
<td>189.2</td>
<td>163.3</td>
<td>148.0</td>
<td>88.4</td>
<td>107.4</td>
</tr>
</tbody>
</table>

TABLE 1 Coverage of cord blood TSH screen – per unit and per year

* b – number of births
* s – number of screened cases
* % - percentage coverage

GSH-Groote Schuur Hospital, MMH-Mowbray Maternity Hospital, NSH-New Somerset Hospital, Vang-Vanguard Drive MOU, Kh-Khayelitsha MOU, Han-Hanover Park MOU, MP-Mitchell’s Plain MOU, Ret-Retreat MOU, Gug-Gugulethu MOU, MOU-Midwife Obstetric Unit
<table>
<thead>
<tr>
<th></th>
<th>2001</th>
<th>2002</th>
<th>2003</th>
<th>2004</th>
</tr>
</thead>
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<tr>
<td>TSH</td>
<td>24690</td>
<td>25982</td>
<td>27129</td>
<td>28173</td>
</tr>
<tr>
<td>tT4</td>
<td>2753</td>
<td>2692</td>
<td>2419</td>
<td>2794</td>
</tr>
<tr>
<td>%</td>
<td>11.1</td>
<td>10.4</td>
<td>8.9</td>
<td>9.9</td>
</tr>
</tbody>
</table>

TABLE 2 Percentage of initial TSH screens requiring tT4 secondary screens.

tT4 numbers not available for 2000

TSH-Thyroid Stimulating Hormone, tT4-total Thyroxine
<table>
<thead>
<tr>
<th></th>
<th>Sex</th>
<th>Birth Weight</th>
<th>Screen TSH</th>
<th>Screen fT4</th>
<th>Repeat TSH</th>
<th>Repeat fT4</th>
<th>Diagnosis</th>
<th>Review age</th>
<th>Start treatment age</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>2.65</td>
<td>51</td>
<td>78</td>
<td>13.2</td>
<td>13.9</td>
<td>Hypoplasia</td>
<td>30d</td>
<td>14 m</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>3.2</td>
<td>80</td>
<td>106</td>
<td>29.1</td>
<td>13.3</td>
<td>Ectopia, Hypoplasia</td>
<td>44d</td>
<td>113d</td>
</tr>
<tr>
<td>3*</td>
<td>M</td>
<td>3.3</td>
<td>57</td>
<td>147</td>
<td>17.0</td>
<td>6.7</td>
<td>Unknown</td>
<td>92d</td>
<td>DNA</td>
</tr>
<tr>
<td>4*</td>
<td>M</td>
<td>1.19</td>
<td>Not doc</td>
<td>Not doc</td>
<td>9.27</td>
<td>15.6</td>
<td>Possible uptake defect</td>
<td>2m</td>
<td>7m</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>3.00</td>
<td>&gt;80</td>
<td>111</td>
<td>29</td>
<td>12.8</td>
<td>Sublingual gland</td>
<td>7m</td>
<td>10m</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>3.5</td>
<td>115</td>
<td>70</td>
<td>&gt;150</td>
<td>7.2</td>
<td>Hypoplasia</td>
<td>55d</td>
<td>72d</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>3.3</td>
<td>51</td>
<td>105</td>
<td>11.28</td>
<td>12.2</td>
<td>Dyshormono genesis</td>
<td>5w</td>
<td>18m</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>3.4</td>
<td>Not doc</td>
<td>Not doc</td>
<td>&gt;150</td>
<td>10.9</td>
<td>Dyshormono genesis</td>
<td>13d</td>
<td>13d</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>1.68</td>
<td>&lt;0.5</td>
<td>71</td>
<td>1.25</td>
<td>5.7</td>
<td>Mat Graves, CMZ</td>
<td>21d</td>
<td>24d</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>2.23</td>
<td>&gt;90</td>
<td>82</td>
<td>&gt;150</td>
<td>4.8</td>
<td>Ectopia, hypoplasia</td>
<td>28d</td>
<td>35d</td>
</tr>
<tr>
<td>11*</td>
<td>F</td>
<td>3.00</td>
<td>53</td>
<td>96</td>
<td>3.69</td>
<td>9.9</td>
<td>Unknown</td>
<td>78d</td>
<td>DNA</td>
</tr>
<tr>
<td>12*</td>
<td>F</td>
<td>2.98</td>
<td>29</td>
<td>88</td>
<td>11.92</td>
<td>14.1</td>
<td>Unknown</td>
<td>54d</td>
<td>DNA</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>0.96</td>
<td>42</td>
<td>69</td>
<td>insuff</td>
<td>7.9</td>
<td>Aplasia</td>
<td>21d</td>
<td>43d</td>
</tr>
</tbody>
</table>

**TABLE 3 Cases of Hypothyroidism diagnosed by screening**

DNA—did not attend follow up, d-day/s, m-month/s, w-week/s,

Mat-maternal, CMZ-Carbimazole

Not doc—not documented, insuff-insufficient

TSH-Thyroid Stimulating Hormone, in mIU/l

fT4-total Thyroxine, in pmol/l

fT4-free Thyroxine, in pmol/l
<table>
<thead>
<tr>
<th>Pt</th>
<th>Sex</th>
<th>Birth weight</th>
<th>Dx</th>
<th>TSH</th>
<th>T4</th>
<th>Start treatment Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>3.96</td>
<td>Sublingual gland</td>
<td>&gt;100</td>
<td>9.1</td>
<td>3wks</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>0.80</td>
<td>Sublingual gland</td>
<td>82.9</td>
<td>6.9</td>
<td>2yr 5m</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>2.57</td>
<td>Aplasia</td>
<td>&gt;100</td>
<td>&lt;2.5</td>
<td>2m 2wks</td>
</tr>
</tbody>
</table>

TABLE 4 Cases of Hypothyroidism missed due to birth outside of screening area.

TSH-Thyroid Stimulating Hormone, in mIU/l

tT4-total Thyroxine, in pmol/l

m-month/s, w-week/s, yr-year/s
<table>
<thead>
<tr>
<th></th>
<th>RCCH</th>
<th>RMOU</th>
<th>MPMOU</th>
<th>All</th>
<th>Hypothyroid cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average</td>
<td>64d</td>
<td>58d</td>
<td>57d</td>
<td>62d</td>
<td>57d</td>
</tr>
<tr>
<td>Mode</td>
<td>45d</td>
<td>46d</td>
<td>51d</td>
<td>40d</td>
<td>21d</td>
</tr>
<tr>
<td>Median</td>
<td>51d</td>
<td>56d</td>
<td>53d</td>
<td>52d</td>
<td>44d</td>
</tr>
</tbody>
</table>

TABLE 5 Age at first review.

MPMOU—Mitchell’s Plain MOU, RMOU—Retreat MOU, MOU—Midwife Obstetric Unit, RCCH—Red Cross Children’s Hospital
d-day/s
<table>
<thead>
<tr>
<th></th>
<th>Sex</th>
<th>Birth Weight</th>
<th>Diagnosis</th>
<th>Review age</th>
<th>Start treatment age</th>
<th>Formal neurodevelopmental assessment</th>
<th>Neurodevelopment comment in notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>F</td>
<td>2.65</td>
<td>Hypoplasia</td>
<td>30d</td>
<td>14 m</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>3.2</td>
<td>Ectopia, Hypoplasia</td>
<td>44d</td>
<td>113d</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>M</td>
<td>3.3</td>
<td>Unknown</td>
<td>92d</td>
<td>DNA</td>
<td>DNA</td>
<td>DNA</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>1.19</td>
<td>Possible uptake defect</td>
<td>2m</td>
<td>7m</td>
<td>DNA</td>
<td>DNA</td>
</tr>
<tr>
<td>5</td>
<td>F</td>
<td>3.00</td>
<td>Sublingual gland</td>
<td>7m</td>
<td>10m</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>3.5</td>
<td>Hypoplasia</td>
<td>55d</td>
<td>72d</td>
<td>Yes, normal at 22m</td>
<td>Normal</td>
</tr>
<tr>
<td>7</td>
<td>F</td>
<td>3.3</td>
<td>Dysthromogenesis</td>
<td>5w</td>
<td>18m</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>3.4</td>
<td>Dysthromogenesis</td>
<td>13d</td>
<td>13d</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>1.68</td>
<td>Mat graves, CMZ</td>
<td>21d</td>
<td>24d</td>
<td>No</td>
<td>Normal on discharge</td>
</tr>
<tr>
<td>10</td>
<td>F</td>
<td>2.23</td>
<td>Ectopia, hypoplasia</td>
<td>28d</td>
<td>35d</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>11</td>
<td>F</td>
<td>3.00</td>
<td>Unknown</td>
<td>78d</td>
<td>DNA</td>
<td>DNA</td>
<td>DNA</td>
</tr>
<tr>
<td>12</td>
<td>F</td>
<td>2.98</td>
<td>Unknown</td>
<td>54d</td>
<td>DNA</td>
<td>DNA</td>
<td>DNA</td>
</tr>
<tr>
<td>13</td>
<td>F</td>
<td>0.96</td>
<td>Aplasia</td>
<td>21d</td>
<td>43d</td>
<td>Yes, normal at 12m</td>
<td>Normal</td>
</tr>
<tr>
<td>14</td>
<td>F</td>
<td>3.96</td>
<td>Sublingual gland</td>
<td>N/A</td>
<td>3wks</td>
<td>No</td>
<td>Normal</td>
</tr>
<tr>
<td>15</td>
<td>F</td>
<td>0.80</td>
<td>Sublingual gland</td>
<td>N/A</td>
<td>2yr 5m</td>
<td>Yes, global delay</td>
<td>Global delay</td>
</tr>
<tr>
<td>16</td>
<td>F</td>
<td>2.57</td>
<td>Aplasia</td>
<td>N/A</td>
<td>2m 2wks</td>
<td>Yes, but record lost</td>
<td>Global delay</td>
</tr>
</tbody>
</table>

**TABLE 6 Neurodevelopmental outcome**

Cases 1-13 Hypothyroidism diagnosed by screening, Cases 14-16 Hypothyroidism missed due to birth outside of screening area

DNA-did not attend follow up, d-day/s, m-month/s, wks-weeks, yr-year/s

N/A-not applicable, DQ-developmental quotient
9. ANNEXURES

ANNEXURE 1

PD 274

CORD BLOOD THYROID SCREEN

GSH/001/PRH/000/000

Key: cl: 166

Mother's Surname: 
Name: 
Folder No: 

DATE OF BIRTH: 29. 11. 2000
BIRTH MASS: 3700

RESULTS:

T4 = 
TSH = 
ANNEXURE 2

Product information on TSH MAIACLONE from BioChem ImmunoSystems

Range:
0.02-50 μIU/ml undiluted, up to 1000 μIU/ml diluted.

Analytical Sensitivity:
0.02-0.04 μIU/ml, the latter when the tracer was at the end of its shelf-life.

Specificity:

<table>
<thead>
<tr>
<th>Analyte</th>
<th>Concentration</th>
<th>Apparent TSH value (μIU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HCG</td>
<td>300 IU/ml</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>200 IU/ml</td>
<td>0.04</td>
</tr>
<tr>
<td></td>
<td>100 IU/ml</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>50 IU/ml</td>
<td>0.06</td>
</tr>
<tr>
<td>FSH</td>
<td>1000 mIU/ml</td>
<td>0.67</td>
</tr>
<tr>
<td></td>
<td>500 mIU/ml</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>250 mIU/ml</td>
<td>0.17</td>
</tr>
<tr>
<td>LH*</td>
<td>1000 mIU/ml</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td>500 mIU/ml</td>
<td>2.3</td>
</tr>
<tr>
<td></td>
<td>250 mIU/ml</td>
<td>1.1</td>
</tr>
</tbody>
</table>

* Endogenous TSH contamination
ND: not detectable

Accuracy:

Recovery experiments were performed by adding purified TSH to pooled serum samples. TSH values were determined before and after addition, and the percentage of recovery of added TSH was calculated.
<table>
<thead>
<tr>
<th>Sample</th>
<th>TSH Added (µIU/ml)</th>
<th>TSH Measured (µIU/ml)</th>
<th>% Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
<td>0</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>0.97</td>
<td>3.48</td>
<td>91</td>
</tr>
<tr>
<td></td>
<td>2.67</td>
<td>5.4</td>
<td>105</td>
</tr>
<tr>
<td></td>
<td>8.84</td>
<td>12.0</td>
<td>106</td>
</tr>
<tr>
<td></td>
<td>26.8</td>
<td>30.6</td>
<td>104</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
<td>1.97</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>0.97</td>
<td>2.93</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td>2.67</td>
<td>4.69</td>
<td>102</td>
</tr>
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<td></td>
<td>8.84</td>
<td>11.2</td>
<td>104</td>
</tr>
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<td></td>
<td>26.8</td>
<td>29.7</td>
<td>103</td>
</tr>
<tr>
<td>3</td>
<td>0</td>
<td>1.45</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td>0.97</td>
<td>2.45</td>
<td>103</td>
</tr>
<tr>
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<td>2.67</td>
<td>4.1</td>
<td>99</td>
</tr>
<tr>
<td></td>
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<td>98</td>
</tr>
<tr>
<td></td>
<td>26.8</td>
<td>28.0</td>
<td>99</td>
</tr>
</tbody>
</table>

Coefficient of variation (CV):

Intraassay Precision (µg/dL)

<table>
<thead>
<tr>
<th>Mean</th>
<th>SD</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.13</td>
<td>3.2%</td>
</tr>
<tr>
<td>2</td>
<td>1.9</td>
<td>3.1%</td>
</tr>
<tr>
<td>3</td>
<td>7.6</td>
<td>3.8%</td>
</tr>
<tr>
<td>4</td>
<td>40.9</td>
<td>3.9%</td>
</tr>
</tbody>
</table>

Interassay Precision (µg/dL)

<table>
<thead>
<tr>
<th>Mean</th>
<th>SD</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.16</td>
<td>3.1%</td>
</tr>
<tr>
<td>2</td>
<td>1.9</td>
<td>2.6%</td>
</tr>
<tr>
<td>3</td>
<td>7.5</td>
<td>1.6%</td>
</tr>
<tr>
<td>4</td>
<td>40.8</td>
<td>2.6%</td>
</tr>
</tbody>
</table>
Product information on Total T4 Cost-A-Count from Diagnostics Products Corporation.

Calibration Range:

1-24 μg/dL. (conversion factor μg/dL × 12.87 = nmol/l)

12.9-309 nmol/l

Analytical Sensitivity:

0.25 μg/dL

3.22 nmol/l

Specificity:

<table>
<thead>
<tr>
<th>Compound</th>
<th>µg/dL Added</th>
<th>% Cross reactivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>L-Thyroxine (T4)</td>
<td>25</td>
<td>100%</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>100%</td>
</tr>
<tr>
<td>D-Thyroxine</td>
<td>10</td>
<td>64%</td>
</tr>
<tr>
<td>Tetraiodothyroacetic acid</td>
<td>10</td>
<td>104%</td>
</tr>
<tr>
<td>Triiodo-L-thyronine</td>
<td>100</td>
<td>2%</td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>ND</td>
</tr>
<tr>
<td>Triiodo-D-thyronine</td>
<td>1</td>
<td>ND</td>
</tr>
<tr>
<td>Triiodothyroacetic acid</td>
<td>1,000</td>
<td>2%</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>ND</td>
</tr>
<tr>
<td>Monoiodotyrosine</td>
<td>1,000</td>
<td>ND</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>ND</td>
</tr>
<tr>
<td>Diiodo-L-tyrosine</td>
<td>1,000</td>
<td>ND</td>
</tr>
<tr>
<td>Methimazole</td>
<td>1,000</td>
<td>ND</td>
</tr>
<tr>
<td>5,5’-Diphenylhydantoin</td>
<td>1,000</td>
<td>ND</td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td>1,000</td>
<td>ND</td>
</tr>
<tr>
<td>6-n-Propyl-2-thiouracil</td>
<td>1,000</td>
<td>ND</td>
</tr>
</tbody>
</table>

ND: not detectable
Coefficient of variation (CV):

Intraassay Precision (µg/dL)

<table>
<thead>
<tr>
<th>Mean</th>
<th>SD</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.4</td>
<td>0.09</td>
</tr>
<tr>
<td>2</td>
<td>4.5</td>
<td>0.15</td>
</tr>
<tr>
<td>3</td>
<td>7.4</td>
<td>0.20</td>
</tr>
<tr>
<td>4</td>
<td>13.8</td>
<td>0.38</td>
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</table>

Interassay Precision (µg/dL)

<table>
<thead>
<tr>
<th>Mean</th>
<th>SD</th>
<th>CV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2.3</td>
<td>0.33</td>
</tr>
<tr>
<td>2</td>
<td>7.2</td>
<td>0.58</td>
</tr>
<tr>
<td>3</td>
<td>11.4</td>
<td>4.8</td>
</tr>
<tr>
<td>4</td>
<td>13.2</td>
<td>0.78</td>
</tr>
</tbody>
</table>

End-of-Run Effect (µg/dL)

<table>
<thead>
<tr>
<th>Tubes</th>
<th>Tubes</th>
<th>Tubes</th>
<th>Tubes</th>
<th>Tubes</th>
<th>Tubes</th>
<th>Tubes</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-48</td>
<td>89-102</td>
<td>143-156</td>
<td>197-210</td>
<td>251-264</td>
<td>305-318</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>2.6</td>
<td>2.4</td>
<td>2.5</td>
<td>2.3</td>
<td>2.3</td>
<td>2.3</td>
</tr>
<tr>
<td>2</td>
<td>4.3</td>
<td>4.2</td>
<td>4.5</td>
<td>4.3</td>
<td>4.6</td>
<td>4.4</td>
</tr>
<tr>
<td>3</td>
<td>8.1</td>
<td>8.1</td>
<td>8.4</td>
<td>8.0</td>
<td>8.0</td>
<td>8.0</td>
</tr>
<tr>
<td>4</td>
<td>11.4</td>
<td>11.5</td>
<td>12.4</td>
<td>12.2</td>
<td>12.3</td>
<td>11.1</td>
</tr>
<tr>
<td>5</td>
<td>14.3</td>
<td>13.9</td>
<td>13.9</td>
<td>14.1</td>
<td>13.9</td>
<td>13.4</td>
</tr>
</tbody>
</table>

This information is provided by the test kit manufacturers, and is published in the package insert in each box of test kits.

The laboratory at RCCH runs its own CVs on every assay. The laboratory performs each test in duplicate. In the rare event of a TSH test falling outside of that assay’s CV, the test is repeated again and a tT4 also run. If there is limited serum, only a tT4 is run. If the sample again fails, or if there is insufficient serum to repeat the test, the result is recorded as a possible abnormality and the laboratory flags the result as requiring recall.
Dear Parent,

All newborn babies born in the Peninsula Maternity Services have a blood test to exclude a deficiency of thyroid hormone. (A substance in the blood which is vital for normal growth and development of your baby).

This test has shown that your baby could be low in thyroid hormone and needs a repeat blood test.

An appointment has been made for your baby at the New Borns Parent Building ...

- No Hospital folder is required
- No Fee is payable
- Bring this letter, your baby and baby's Clinic Card with you
- If you cannot make the above appointment please come on the next Monday at 8am

Please note that the doctor will not be present at any other time during the week to see your baby.

Please show this letter to the Reception Clerk.

Yours faithfully,

Sr V L Sterck

DOB 7/4/01

Red Cross War Memorial Children's Hospital
Private Bag
Rondebosch
7701

Tel: (021) 659-5111
Fax: (021) 683-3801

T - "Hospital"
14 January 2005

REC REF: 024/2005

Dr IAM Carrihill
Paediatric Endocrinology
Ward 625
NGSH

Dear Dr Carrihill

AN AUDIT OF THE THYROID SCREENING PROGRAMME IN THE PENINSULA MATERNAL
AND NEONATAL SERVICES

Thank you for submitting your study to the Research Ethics Committee for
review.

It is a pleasure to inform you that the Ethics Committee has formally approved
the above-mentioned study on the 12 January 2005.

Please note that any ethical consideration such as confidentiality and anonymity
of information remain the responsibility of the investigator.

Please quote the REC. REF in all your correspondence

Yours sincerely

PROF T. ZABOW
CHAIRPERSON
10. REFERENCES


7. Boileau P, Bain P, Rives S, Toublanc J.E. Earlier onset of treatment or increment in LT4 dose in screened congenital hypothyroidism: which as the more important factor for IQ at 7 years? Horm Res. 2004;61(5): 228-33


24. Maughan K. Parents win R100m claim for girl’s disability. Cape Argus 2004 Feb 25;1
25. Schroeder F. Court cuts doctor’s blame for brain damage. Cape Times 2006 Sep 07;4


11. ACKNOWLEDGEMENTS

Dr S.V. Delport, Head of Paediatric Endocrinology, School of Child and Adolescent Health, UCT

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Dr D. Greenfield, Community Neonatology, PMNS

Dr T. van Heerden, Community Neonatology, PMNS

Dr M. Kroon, Neonatology Consultant, MMH

Dr V. Ramanjum, Neurodevelopmental Unit Consultant, CHI

Lizette Berry, Child Poverty Programme, CHI

Sue Philpott, Disability task team for the Children’s Bill process

RCCH Endocrine clinic staff: Sr V.L. Starck, Sr A. Went, Sr J. Turner and volunteers: Pam Klerk and Lillian Bosman

Laboratory technologists: Carin Verburg, Glenda Fenemore and Sue Lindsey

Thank you all. Your ongoing actions have kept the screening programme alive, and allowed this audit to be achieved.