Short and Tall Stature

Dr Wayne Cutfield

The following is an actual question given on a University of Washington engineering midterm.
Bonus Question: Is Hell exothermic (gives off heat) or Endothermic (absorbs heat)? Most of the students wrote Proofs of their beliefs using Boyle's Law, (gas cools off when it expands and heats when it is compressed) or some variant.

One student, however, wrote the following: "First, we need to know how the mass of Hell is changing in time. So we need to know the rate that souls are moving into Hell and the rate they are leaving. I think that we can safely assume that once a soul gets to Hell, it will not leave. Therefore, no souls are leaving. As for how many souls are entering Hell, let us look at the different religions that exist in the world today. Some of these religions state that if you are not a member of their religion, you will go to Hell.

This gives two possibilities: 1. If Hell is expanding at a slower rate than the rate at which souls enter Hell, then the temperature and pressure in Hell will increase until all Hell breaks loose. 2. Of course, if Hell is expanding at a rate faster than the increase of souls in Hell, then the temperature and pressure will drop until Hell freezes over.

So which is it? If we accept the postulate given to me by Teresa Banyan during my Freshman year, "...that it will be a cold day in Hell before I sleep with you.", and take into account the fact that I still have not succeeded in having sexual relations with her, then, #2 cannot be true, and thus I am sure that Hell is exothermic and will not freeze." This student received the only A.

Since there are more than one of these religions and since people do not belong to more than one religion, we can project that all souls go to Hell. With birth and death rates as they are, we can expect the number of souls in Hell to increase exponentially. Now, we look at the rate of change of the volume in Hell because Boyle's Law states that in order for the temperature and pressure in Hell to stay the same, the volume of Hell has to expand as souls are added.
Short stature and tall stature are symptoms or signs **NOT** diseases nor a diagnosis.

### Normal Growth

**REQUIREMENTS for growth/ anabolism**
- Nurturing, caring environment
- Adequate nutritional supply and the ability to digest and absorb the food (ie the child should be well nourished)
- Appropriate hormonal milieu
- Appropriate extracellular and intracellular environment.

### Definitions

<table>
<thead>
<tr>
<th>Term</th>
<th>Description</th>
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<tbody>
<tr>
<td>Short stature</td>
<td>Height &lt;3rd PC</td>
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<tr>
<td>Dwarfism</td>
<td>Ht &gt;3 SD below mean for age.</td>
</tr>
<tr>
<td>Tall stature</td>
<td>height &gt;97th PC</td>
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Can calculate velocity from 2 heights (cm/yr) or 2 relative heights (Ht SDS-Ht SDS: 0 SDS/yr normal).

### Short Stature Presentation

In excess of 80% are boys. Presentation when starting primary school or high school. Although 95% of children with short stature are normal variants, short stature is one of the most useful indicies of undiagnosed chronic illness.

### Short Stature History

Antenatal history (drugs, illnesses). Birth weight and gestation. Feeding pattern (beware failure to thrive). Thorough systems enquiry (particularly renal and GI symptoms). Hospital admissions and drug history (beware steroids, inhaled, topical, oral) Social history (psychosocial dwarfism is not exclusive to families of lower social class).

### Short Stature History

Mother and fathers heights. MPH = M(cm) + F (cm) ±13 cm /2. MPH range ±8 cm. FH short stature: males <165 cm females <152 cm FH delayed puberty: menarche >14 yrs in females and continued growth after high school in males. Look at other sibs child development records.
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Look at other sibs child development records.

### Child Development record

A valuable source of information.
Look at all available height and weight measurements and growth trend.
Remember that Plunket height measurements are not precise and may be misleading.
Check developmental milestones and illnesses.

### Short Stature Examination

Measure height ACCURATELY.
Height can only be measured accurately with a stadiometer. Pharmacia Corporation sell them to GPs.
Correct height measurement technique imperative in calculation of height velocity.
Shoes off, flat footed, extend neck, head in neutral plane. Mean of 3 measurements.

### Short Stature Examination

Span and sitting height most accurate indicies of disproportionate short stature.
Lower to upper segment measurement very inaccurate.
Weigh lightly clothed.
Dysmorphic features.
Pulse rate and BP.
Visual fields and fundi.
Assess pubertal development (testicular vol >3 cm in males, Tanner 2 breast development in females.

### Plotting growth

The Adelaide Growth charts are the most comprehensive available and contain ht, wt, HV, pubertal staging and timing, HC, neonatal Lt and wt charts, stretched penile lengths, BSA formula.
Plot height and weight measurements.
Transpose MPH and MPH range as “targets” for growth.

### Failure to thrive

If weight deviates further below the 3rd percentile than height the problem is failure to thrive (or failure to gain weight).
Weight usually deviates below the 3rd PC before height.
Often presents within the first year of life.
If in doubt calculate Ht and Wt SDS.
Do not confuse with short stature.
Height velocity (HV)

If history and examination do not point to an underlying chronic illness and the child is not >5 cm below 3rd PC Height velocity is the most sensitive index to remeasure height in 6-12 months.
HV differentiates normal variant short stature from pathological short stature.
HV cannot be calculated over shorter interval because of errors in measurement.
Normal height velocity 25-75 PC.
Note HV curve for children with delayed puberty.

Normal Variant Short Stature

Familial short stature.
Constitutional delay of growth and development.
Account for >95% of children who present with short stature.

Normal Variant Short Stature

<table>
<thead>
<tr>
<th>Birthweight</th>
<th>FSS</th>
<th>CDGD</th>
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<tbody>
<tr>
<td>Normal</td>
<td></td>
<td>Normal</td>
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<table>
<thead>
<tr>
<th>Chronic illness</th>
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<th>CDGD</th>
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<tbody>
<tr>
<td>Absent</td>
<td></td>
<td>Absent</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Family history</th>
<th>FSS</th>
<th>CDGD</th>
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</thead>
<tbody>
<tr>
<td>FSS</td>
<td></td>
<td>CDGD</td>
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</table>

<table>
<thead>
<tr>
<th>Infant growth</th>
<th>X centiles</th>
<th>X centiles</th>
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<table>
<thead>
<tr>
<th>Childhood HV</th>
<th>Normal</th>
<th>Normal</th>
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| Late childhood HV | normal | slow |

<table>
<thead>
<tr>
<th>Bone Age</th>
<th>FSS</th>
<th>CDGD</th>
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</thead>
<tbody>
<tr>
<td>&lt;1 yr from CA</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>&gt;1 yr from CA</td>
<td></td>
<td></td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Puberty</th>
<th>FSS</th>
<th>CDGD</th>
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</thead>
<tbody>
<tr>
<td>On time</td>
<td></td>
<td>Delayed</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Final Height</th>
<th>FSS</th>
<th>CDGD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Short</td>
<td></td>
<td>Normal</td>
</tr>
</tbody>
</table>

Bone Age

Gruelich and Pyle standards.
Imprecise picture matching.
1 yr intervals.
Tables of Bayley and Pinneau for final Ht prediction.
PAH very useful to distinguish FSS and CDGD for Dx and reassurance.

Poor Height Velocity

Assume pathological short stature until proven otherwise.
Screening Ix: FBC, ESR
LFTs, U and Es, Cr, Ca.
Urinalysis and culture.
Karyotype (F).
TFTs.
Bone age.
Random GH meaningless.
### Pathological short stature

<table>
<thead>
<tr>
<th>Proportionate:</th>
<th>IUGR syndromes</th>
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<tbody>
<tr>
<td></td>
<td>chronic illness</td>
</tr>
<tr>
<td></td>
<td>drugs</td>
</tr>
<tr>
<td></td>
<td>psychosocial deprivation</td>
</tr>
<tr>
<td>Disproportionate:</td>
<td>Syndromes (partic Turner S)</td>
</tr>
<tr>
<td></td>
<td>hypothyroidism</td>
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<tr>
<td></td>
<td>Skeletal dysplasias</td>
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</table>

### IUGR

Very common.  
Birth weight <10th PC for gestational age.  
Catch-up growth above 3rd PC usually occurs by 6 mos of age but may drag on to 2 yrs.  
Short stature by 2 yrs usually associated with short final height.  
As a group these children do not reach their MPHs.  
Approx 10 PC become short adults.  
Often associated with CDGD.

### Turner Syndrome

Consider in all girls with unexplained short stature or Ht below MPH range.  
Commonest feature is short for MPH (100%).  
Hyperconvex nails strong clue.  
50% will only have short stature as clinical feature.  
Present with short stature or delayed puberty.  
GH has been shown to improve final Ht.  
Sex steroid therapy needed at puberty.  
Refer to Paed Endocrinologist when Dx made.

### Skeletal dysplasias

Not always obvious.  
Consider if:  
marked familial short stature present.  
BA not delayed and Ht PC well below MPH range.  
Check sitting Ht and span.  
Skeletal survey may be helpful.

### Tall Stature Presentation

In excess of 80% are girls.  
Presentation when starting primary school or high school.  
95% of children with tall stature are normal variants.

### Tall Stature History

Birth weight.  
Developmental milestones.  
Careful systems enquiry particularly: visual, cardiac, joint, learning, coordination problems, headaches or chronic vomiting.  
Parent and sib heights.  
Parents weights and age of puberty.
### Tall Stature Examination

Accurate Ht, Wt, span, sitting Ht, HC.
Who does this child look like.
Arachnodactyly and eunichoid habitus.
Accral changes of acromegaly.
Hyperextensible joints.
Lenses and visual fields.
Pubertal development.

### Normal Variant Tall Stature

Careful history and exam do not indicate an underlying pathological cause.
Most cases of pathological tall stature have dysmorphic features, albeit subtle.
Familial tall stature.
Early maturation.
Exogenous obesity.
Height velocity less helpful at distinguishing normal from pathological tall stature.
Refer if Ht PC above MPH range

### Normal Variant Tall Stature

<table>
<thead>
<tr>
<th>Family Hx</th>
<th>FTS</th>
<th>Early maturat.</th>
<th>Obesity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height vel.</td>
<td>N or ↑</td>
<td>N or ↑</td>
<td>↑</td>
</tr>
<tr>
<td>Dysmorphic exam</td>
<td>No</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Bone age</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Puberty</td>
<td>On time</td>
<td>Early</td>
<td>Early</td>
</tr>
<tr>
<td>Final Ht</td>
<td>Tall</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

### Pathological tall stature

- **Syndromes:** Klinefelter’s
- Marfan’s
- Sotos (cerebral gigantism)
- **Endocrine disorders:** Pituitary gigantism
- Hyperthyroidism
- Precocious puberty
- Cong. Adrenal hyperplasia
- **Others:** Homocysteinuria

### Pathological tall stature Investigation

- Bone age
- CXR
- TFTs
- IGF-1
- Serum 17 hydroxyprogesterone
- Urinary homocysteine
- Cardiac ultrasound
- Ophthalmological review