Clinical assessment of ADHD

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Assessment is more than recognition

- Detecting the disorder
- Contextual assessment
- Recognition of accompanying problems
- Subtyping
- Investigating aetiology
- Understanding the presentation
- Assessing for therapy

The size of a clinic's problem

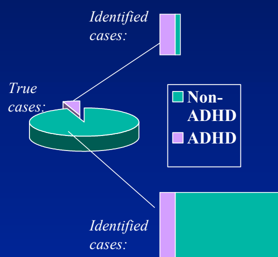
Population	150,000
Children	30,000
No. with ADHD	1,800
Hyperkinetic	450

Detailed multidisciplinary assessment is often impractical: what are the basic standards?

Diagnosis: detecting the disorder

- Screening rating scales:
  Conners, CBCL, DSM-IV,SDQ
- Clinical interview with parent
- Observation:
  consider school obs. if in doubt
- Psychological tests:
  CPT, stop, delay, MFF, FFD, TEACH

95% Sensitivity & Specificity is not good enough



Most cases, identified as ADHD by rating scales, actually come from the non-ADHD population

ADHD has several components

- Inattentiveness
  - disorganised, forgetful, does not invest effort,
  - brief and changing activities
- Overactivity
  - depending on context
- Impulsiveness

Impulsiveness: action without reflection

- Impulsive behaviour style
  - blurting out answers
  - not waiting for one's turn
  - interrupting others
- Impulsiveness as an underlying construct
  - Failure of inhibitory functions
  - Failure of other executive functions
  - Altered motivation

NOT ADHD: "Impulsions" to specific behaviours
Explosive anger
Defiance
Problems with sleep

The behavioural interview

- Length of time on activities
  - modelling,reading,homework,drawing (NOT computer games; TV only for pre-8)
- Movement when calm expected
  - mealtimes,church,visiting,shopping,journeys
- Organisation of activities
  - preparation,memory,detail,listening

Most discriminative items from investigator-based interviews

Clinical observation

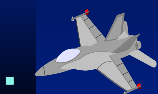
- Preschool
  - Time on task, activity changes
  - Waiting, esp. for reward
  - Social disinhibition
- School age
  - Premature decisions in test situations
  - Difficulty in slowing tempo
  - Lack of reserve and discretion

Novelty and monitoring reduce hyperactivity markedly; first impressions may give false negatives

Cognitive changes in attention deficit?

- Poor performance on "attention tests"?
  - *Slow, variable reaction times, CPT errors*
- Impairments of sustaining or selecting?
  - *do not in fact characterise ADHD*
- Poor executive function?
  - *only some such tests are linked after IQ control*
- Impaired inhibition?

STOP TASK



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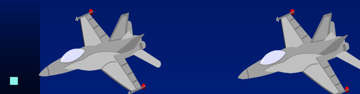
STOP TASK



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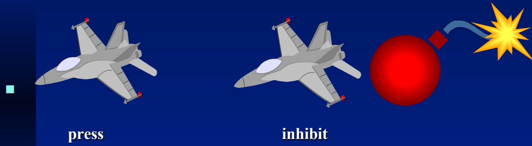
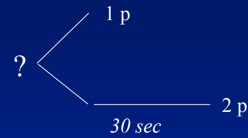



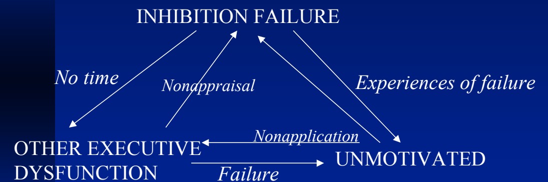
- Inhibition of a planned motor response

Choosing the immediate reward

?
1 p
2 p
30 sec

Reasons for impulsiveness

- Failure of inhibitory functions
  - Cannot inhibit at all/cannot inhibit a prepotent response/cannot wait
- Failure of other executive functions
  - Cannot time correctly/cannot plan/cannot regulate state
- Altered motivation
  - Lack incentive/rapid decay of reward/delay averse/altered cognitive energetics

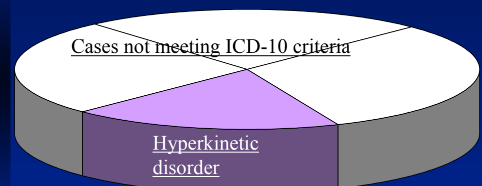


Situational hyperactivity

Different associations

School only	Both	Home only
Poor reading Low achievement	Language delay	Little cognitive impairment
Late onset	Motor clumsiness	Family adversity

Subtypes of AD/HD

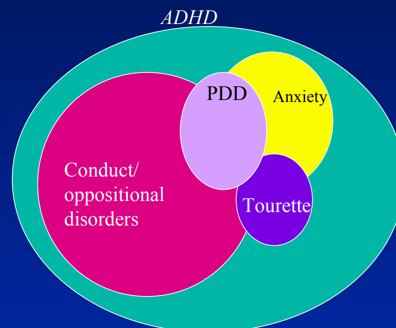


The conclusions from research on the hyperkinetic group do not necessarily apply to other subtypes of ADHD

Hyperkinetic disorders

- Pervasive inattention & impulsiveness
- Absence of affective or PDD comorbidity
- [Early onset of problems]
- Impairments in stopping & delaying
- Language problems, motor incoordination, brain imaging changes
- >90% responsive to stimulants

Other symptom patterns coexist



Two disorders

- HYPERACTIVITY
  - Impulsiveness
  - Inappropriate activity
  - Inattentiveness
- CONDUCT DISORDER
  - Oppositionality
  - Aggression
  - Offending

Two literatures

- HYPERACTIVITY
  - Genetic influences
  - Cognitive deficit
  - Medication response
- CONDUCT DISORDER
  - Social influences
  - Attribution changes
  - Treatment resistance

Similar problems

- HYPERACTIVITY
  - Impulsiveness
  - Inappropriate activity
  - Inattentiveness
- CONDUCT DISORDER
  - Poor impulse control
  - Social rule breaking
  - Academic failure

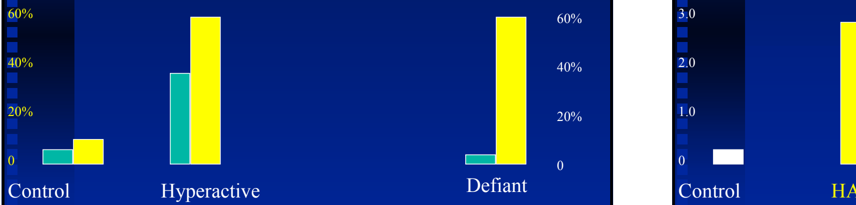
Disentangling HA and CD

Population study in East London

- All 7-year-old boys (3,215) identified from school rolls and health records
- Parent & teacher Rutter scales for 2,462
- Stratified behaviourally into Hyperactive (HA), Defiant, Inattentive, Mixed & Problem-free (Control)
- Random selection of 50 in each group
- Detailed interviews & tests: 91%compliance

Impulsiveness in diffferent groups

- Impulsive test responses at age 7
- Impulsive behaviours in classroom



Cases from a population sample of 2,600; N=45 in each group; stratified by rating scales

Hyperactivity & conduct disorder

- Neuropsychological associations at age 7

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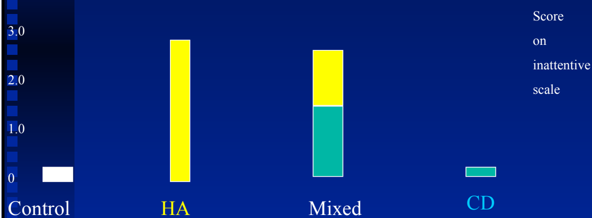


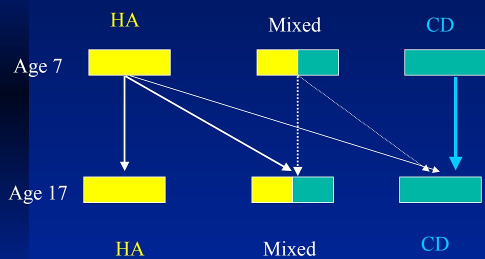
Cases from a population sample of 2,600; N=45 in each group; stratified by rating scales

Outcome measures

- Parental interview ratings
- Psychiatric interview with youths
- Cognitive testing
- Home Office records of offending
- School records

88% follow-up 10 years later; nonresponders similar to responders

Hyperactivity & conduct disorder



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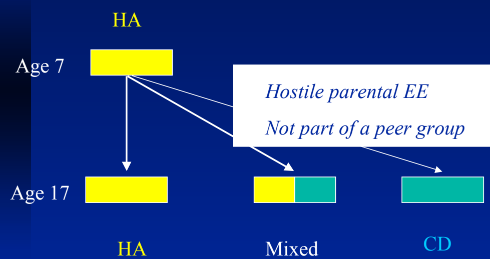
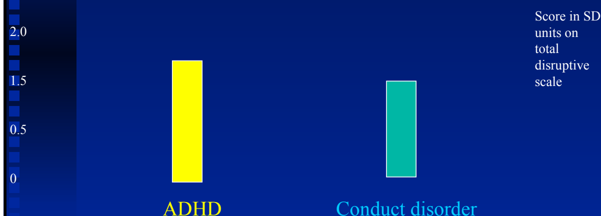


Hyperactivity & conduct disorder

- Effect size of methylphenidate



Cases from a clinic sample of boys referred because of disruptive behaviour: Psychol Med 1987

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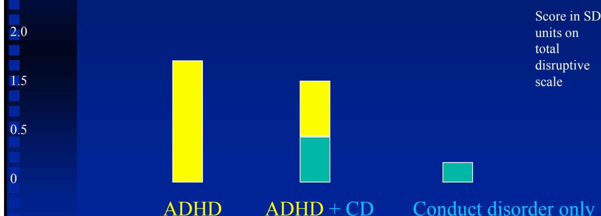


Cases from a clinic sample of boys referred because of disruptive behaviour: Psychol Med 1987

Common types of comorbidity

- Conduct and oppositional disorders
- Anxiety
- PDD
- Tourette
- Epilepsy
- Attachment disorder
- Family adversity

ADHD and Tourette disorder

- Possible common causes
  - Conflicting genetic findings; ?ADHD is not more common in relatives of TD probands, but is more common in relatives who have TD
  - MRI changes in TD include those of ADHD
- Sometimes a iatrogenic link
- “Overactivity” may be multiple tics
- ADHD predicts poor adjustment in TD

ADHD and Anxiety

- Sometimes anxiety results from failure
  - but not found in group studies
- Sometimes “anxiety” means ADHD
  - volatility, overreactiveness, dysregulation
- Sometimes “ADHD” means agitation
- Sometimes anxiogenic environment comes from ADHD parents

Contradictory findings on drug responsiveness and longitudinal course

ADHD and autism

- Sometimes two distinct disorders
  - independent actions of stimulants
  - additive neuropsychological changes
- Sometimes an autistic overactivity
  - hyperkinesis with stereotypies; catatonia
- Sometimes a iatrogenic link
- Sometimes a common cause

ADHD and bipolar disorder

- Criterion overlap
- Controversial redefinition of juvenile mania
  - "mania" without euphoria or cycling course
- Some structured interviews relax criteria
- Possible common causes
  - epilepsy, monoamine changes at synapse, neurodevelopmental antecedents

ADHD and reading problems

- In theory, ADHD and RP could influence each other
- Association is with school ADHD only
- Different cognitive patterns
- After the age of 7 ADHD and RP develop independently
- Association probably due to early genetic & environmental influences

Common types of comorbidity

- Conduct and oppositional disorders
- Anxiety
- PDD
- Tourette
- Epilepsy
- Attachment disorder
- Family adversity

Conclusions about comorbidity

- CD is a mediated complication of ADHD
- Association of ADHD with other syndromic patterns reflects a variety of pathways
- Known genetic and environmental causes are of small effect and often low specificity
- Dimensional and developmental track formulations rather than "comorbidity"
- Definitions are often imprecise

Suggested clinical subgroups

- Hyperkinetic disorder
- Attention deficit without hyperactivity
- School-specific ADHD
- Home-specific disruptiveness
- Comorbid with affective disorder
- Pervasive developmental disorders; attachment disorders; Tourette; et al

But genetic evidence does <u>not</u> support subgrouping

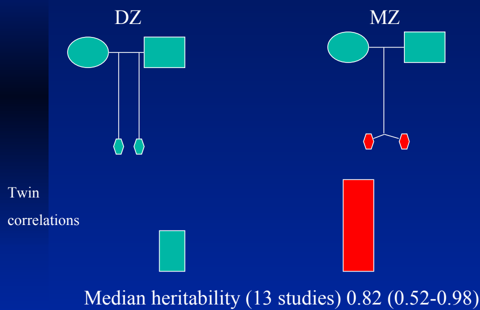
Investigations

- Guided by clinical evaluation
- Always: family life; hearing
- Developmental delay: chromosomes
- Episodic changes: EEG
- School problems: psychometrics
- Deterioration: full CNS evaluation

Most commonly detected: epilepsy, LD, hearing loss, tics, PDD

Occasional: FAS, fraX, chromosomal, Williams, SFS, Pb, TS, etc.

Twin studies show high heritability



Twin correlations

Median heritability (13 studies) 0.82 (0.52-0.98)

What is inherited?

- Not ADHD: genetic influences on continuum*
- Not a unitary trait: influences vary with context
- Dispositions to react:
  - gene-environment interactions and correlations
    - adoptive & longitudinal studies indicate parenting influences
    - MAOA activity influences effect of parenting on aggression
    - pre and perinatal adversity has wide range of expression

*(with possible exception at highest level of severity)

Probable environmental associations

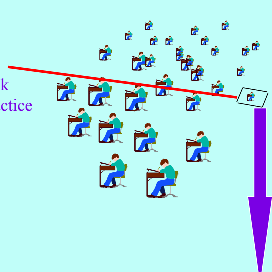
- Physical
  - Lead, alcohol, tobacco & drug exposure of fetus
  - Perinatal: LBW, ?hypoxia, hypoglycaemia
- Early environment
  - Depriving institutions; non-attachment
  - Diet; encephalopathies (incl PANDAS?)
- Intrafamilial
- External stresses on family
- Neighbourhood and school

European Perspectives on Treatment

- Infrequent recognition
  - single-diagnosis schemes; referral filters
- Diagnosed cases are complex & severe
- Economics are of tax-funded care
- CD dominates referral & research
- Psychological approaches preferred to physical; and sometimes group to individual

The route to treatment:

about 1 in 10 of those at risk receive the diagnosis in practice



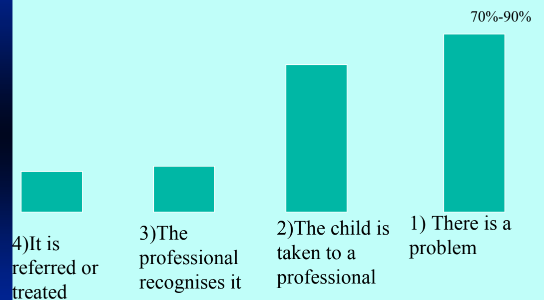
4)It is referred or treated

3)The professional recognises it

2)It is taken to a professional

1) There is a problem

Stages in the career of a child with hyperactive behaviour



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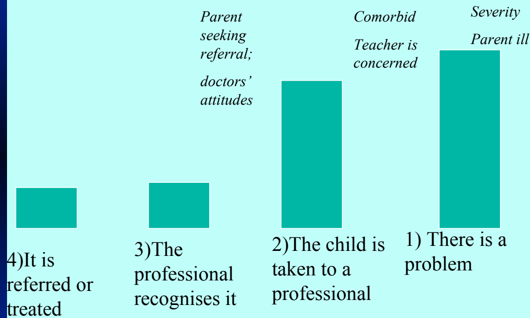
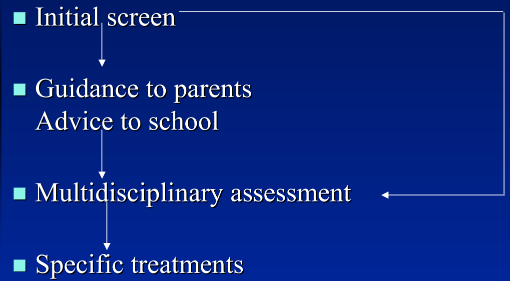


Data from "Filters in referral":Sayal,Byrne,Taylor

Stages of assessment



- Initial screen
- Guidance to parents
  Advice to school
- Multidisciplinary assessment
- Specific treatments

Specific treatments

- Licensed drugs:
  Methylphenidate and dexamphetamine
- Unlicensed drugs:
  Trial evidence: pemoline, imipramine, clonidine, atomoxetine, bupropion, "Adderall", extended-release MP
  Anecdotal: guanfacine, moclobemide, venlafaxine, risperidone
- Psychological therapies:
  Parent training, school liaison, social skills

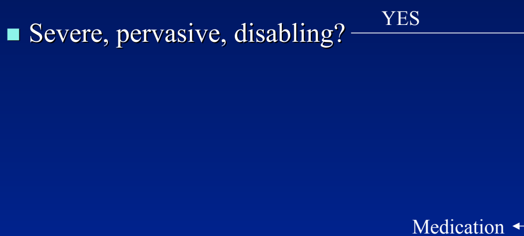
Include non-specific interventions - education, support, advice

Using drugs - a controversy

- Public understanding of medicine
- Ideological battlegrounds
- Inadequate scientific evidence
- Wider social anxieties

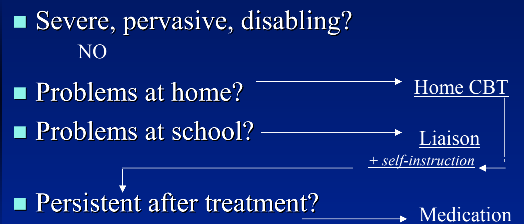
Treatment decisions

- Severe, pervasive, disabling? YES



Treatment decisions

- Severe, pervasive, disabling?
  NO
- Problems at home? → Home CBT
- Problems at school? → Liaison + self-instruction
- Persistent after treatment? → Medication



Treatment decisions

- Severe, pervasive, disabling?
- Problems at home? → Home CBT
- Problems at school? → Liaison
  + self-instruction
- Persistent after treatment? → Medication
- Comorbid problems?

Recommendations

- Many measures from research are best seen as screening instruments in the clinic
- Assessment must be full enough to detect coexisting problems
- ADHD varies with context: no single rule for blending sources; recognise implications; subtype
- ADHD is a description not an explanation; seek contributing influences