Brain Injury Psychiatry Workshop

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Overview- Jodi Cartoon, Fellow

- 10:15-11:15 Frontal Dysregulation Syndromes
  - Apathy, abulia with case presentation
  - Dysregulated, labile mood with case presentation
  - Compulsive behaviours, aggression and sexual disinhibition

- 11:30-12:30 Discrete Psychiatric Conditions
  - Anxiety and depression with case presentation
  - Psychosis with case presentation
  - Obsessive Compulsive Disorder (OCD) and Post Traumatic Stress Disorder (PTSD)

- 13:30-14:30 Workshop

- 14:45-15:45 Panel discussion

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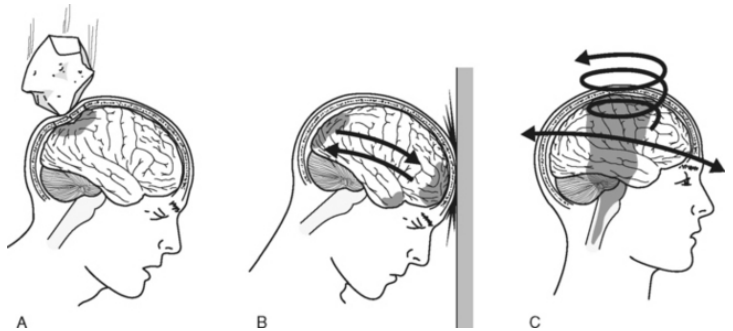
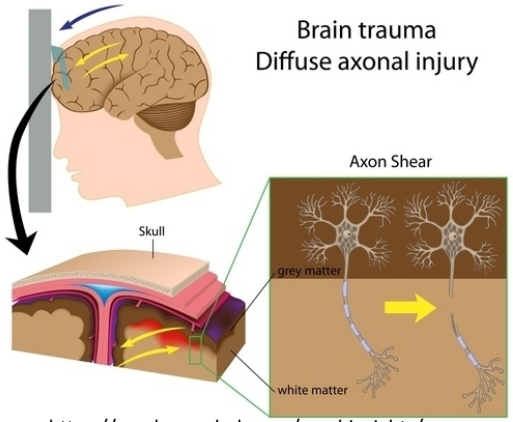
Background

- The neurobiological mechanisms of TBI involve the combination of focal damage and diffuse injury
- Focal and macroscopic- at the time of the event
  - direct insult
  - linear acceleration and deceleration
  - rotational forces

  includes cortical and subcortical lacerations and intracranial bleeding as well as secondary oedema and herniation
- Diffuse and microscopic- pathological stretching and tearing of axons



https://musculoskeletalkey.com/traumatic-brain-injuries/



https://psychscenehub.com/psychinsights/neuropsychiatry-of-traumatic-brain-injury/

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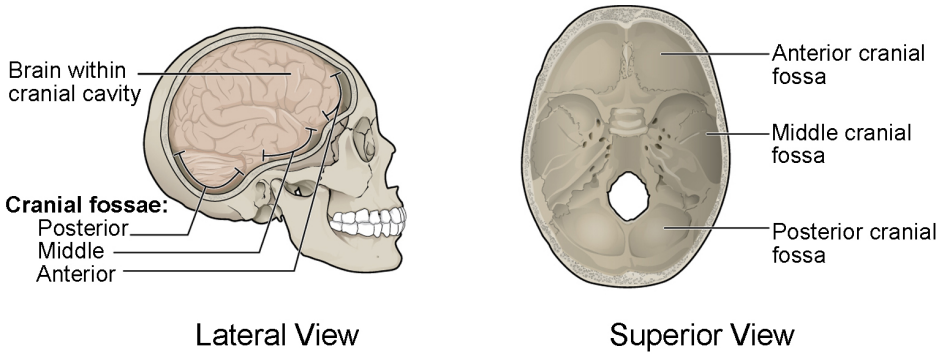
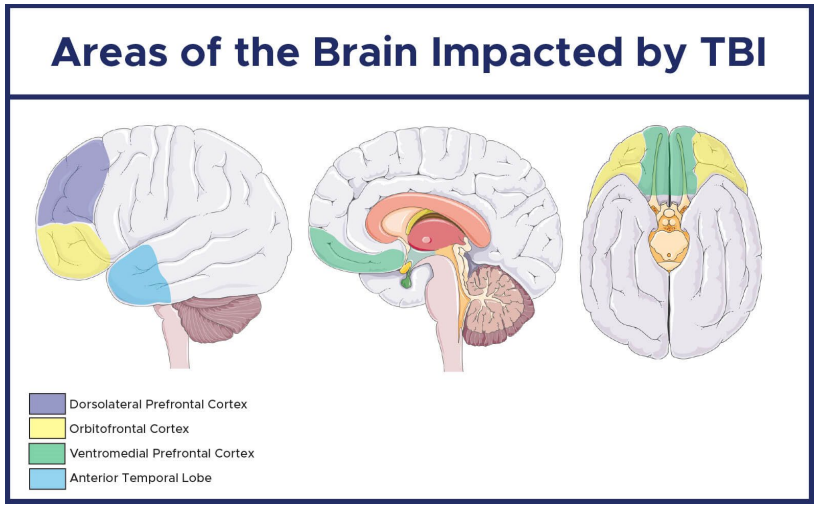
Focal- Predictable pattern of damage

- Frontal and temporal lobes cradled by anterior and middle cranial fossa- irregular surface, numerous bony protuberances
  - Parenchyma → contusions
  - Blood vessel shearing → haemorrhage



Anatomy & Physiology, Connexions Web site. http://cnx.org/content/col11496/1.6/



https://verdugopsych.com/neuropsychological-evaluation-of-traumatic-brain-injury/

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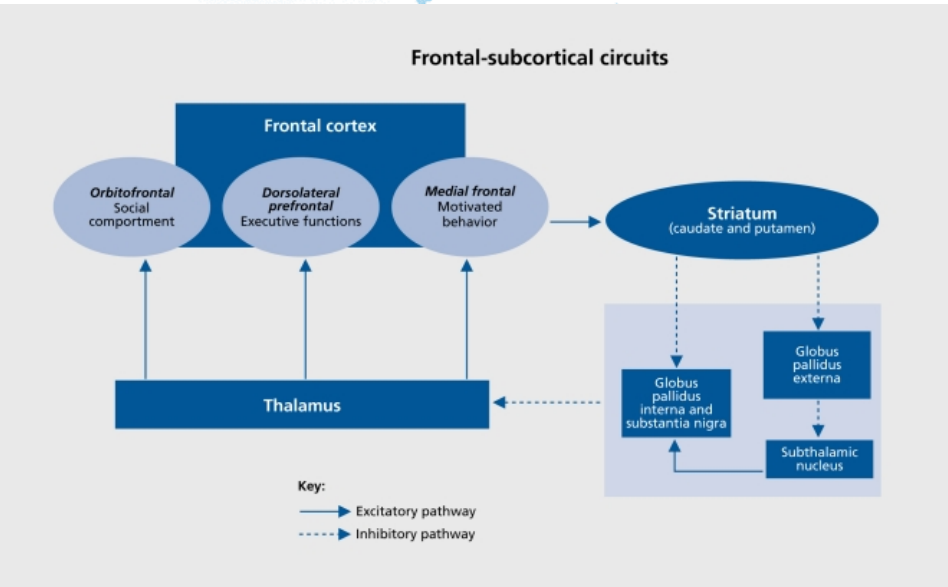
Predictable neuropsychiatric sequelae



Frontal-subcortical circuits- cognition and social behaviour

- **dorsolateral prefrontal cortex-** modulates executive functions: forming and implementing goals and plans (working memory, decision making, problem-solving, and mental flexibility)
- **orbitofrontal/ventral cortex-** personality changes: disinhibited, impulsive, labile, poor insight and reduced concerns for consequences
- **anterior cingulate/medial prefrontal-** goal directed cognition, emotion, behaviour: apathy, lack spontaneous speech and movement

Anterior temporal lobe/temporal pole/paralimbic region- sudden and unexpected bursts of anger, memory impairment for recent events, depression, hallucinations, rapid mood swings, and epileptic seizures

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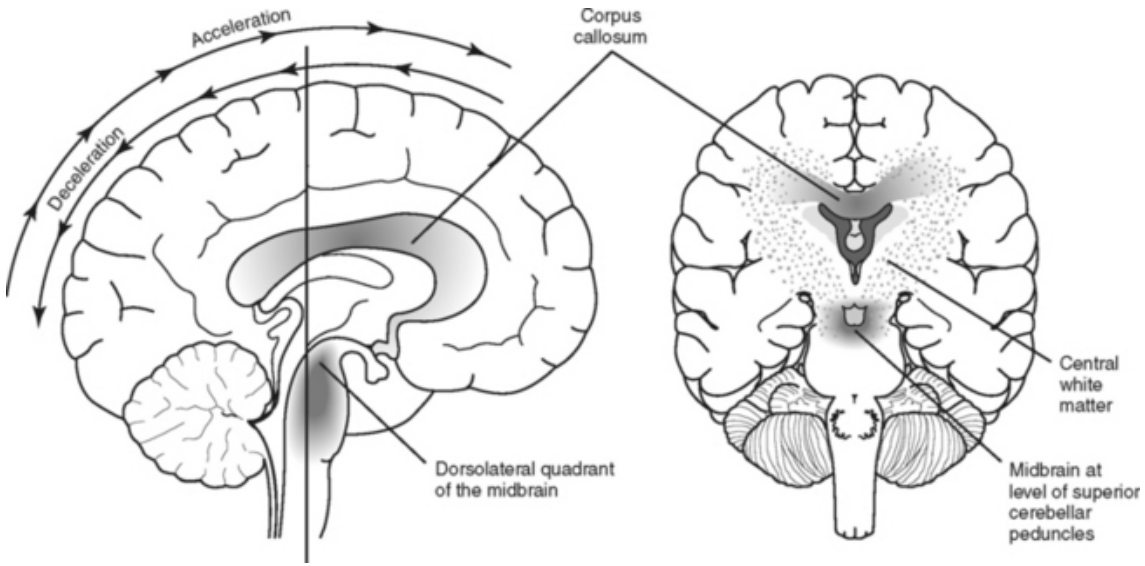
Diffuse- Predictable pattern of damage

- Diffuse axonal injury (DAI)- subcortical white matter throughout the hemispheres, especially at the grey-white matter interface, the corpus callosum, and the brainstem
- Memory and executive dysfunction are the most common neuropsychological impairments found in patients that had CT scan or MRI findings compatible with DAI



https://musculoskeletalkey.com/traumatic-brain-injuries/

Case 1

- 48 yr old married father, CALD background, high powered exec role. Extremely severe TBI (PTA 50 days) 2 yrs prior, MVA v pedestrian. MRI- L. anterior frontal contusions and DAI.
- Physically recovered well aside from some reduced ROM L. upper limb
- Significant ongoing issues with memory, attention, fatigue, concentration and language
  - Carers 6hrs/day, reduced over time
  - OT: Road safety, memory strategies, planning tasks, study options, vocational provider
  - Physio: Shoulder ROM, exercise program
  - Speech: Word retrieval, reading comprehension, auditory comprehension
  - SW: Finances, support for wife
  - Case management: Liaison with carers, relevant financial parties, goal setting

PTA= Posttraumatic amnesia, orientation and memory for ongoing events

Case 1

- Low mood and aggression- seeing psychologist, difficulty accepting change in role. Psychoeducation, cognitive behavioural therapy for adjustment/irritability/mood
- Expressed suicidal ideation- referred to psychiatry clinic
- Hopeless with poor mood, energy, motivation, sleep. Antidepressant commenced which helped with mood fluctuations and anger, melatonin helped with sleep
- Ongoing prominent lack of initiation and drive, spending most of day watching Youtube and not attending study course as “boring”. Lying in bed until compelled to emerge by wife, wouldn’t instigate conversation or activities

Apathy

- Reduction in motivation and goal-directed activities:
  - Behaviours (lack of effort, initiative, and productivity)
    - Emotional concomitants of behaviours (flattened affect, emotional indifference, restricted emotional responses to important life events)
  - Cognitions (decreased interests, lack of plans and goals, lack of concern about one's own personal problems)

Starkstein SE, Leentjens AF: The nosological position of apathy in clinical practice. J Neurol Neurosurg Psychiatry 79(10):1088–1092, 2008

Differentiating apathy

- Can be reversed by external stimulation v pathology of the neuromuscular system
- Overlap with depression- loss of interest and pleasure
- Cognitive deficits may impair pursuit of pastimes and iADLs

Glenn MB, Burke DT, O'Neil-Pirozzi T, et al: Cutoff score on the apathy evaluation scale in subjects with traumatic brain injury. Brain Inj 16(6):509–516, 2002
Starkstein SE, Merello M: Psychiatric and Cognitive Disorders in Parkinson's Disease. Cambridge, UK, Cambridge University Press, 2002

Abulia

- Conceptualised as more severe type of apathy with lack of self-initiation and self-regulation of purposeful behaviour and concern, and severely impaired ability to communicate
- Difficulty initiating and maintaining purposeful movements, poverty of spontaneous movements, reduced spontaneous speech, increased response time, reduced social interaction

Vijayaraghavan L, Krishnamoorthy ES, Brown RG, et al: Abulia: a delphi survey of British neurologists and psychiatrists. Mov Disord 17(5):1052–1057, 2002

Apathy- Proposed mechanism

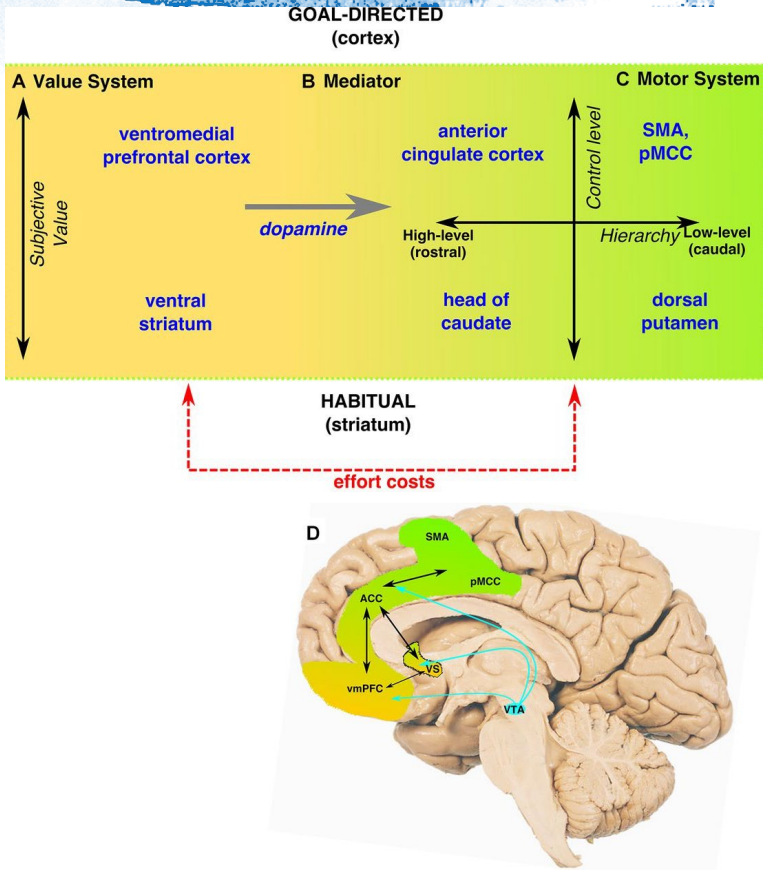
- Le Heron et al- Three fundamental processes
- Premise: importance of outcomes for motivating behaviour - philosopher and physician John Locke ascribed a crucial role for reinforcers (pleasure and pain) for motivating actions, writing that without these perceptions

"… we should have no reason to prefer one thought or action to another … and so we should neither stir our bodies, nor employ our minds, but let our thoughts (if I may so call it) run adrift … In which state man … would be a very idle, inactive creature, and pass his time only in a lazy lethargic dream."

Le Heron C, Holroyd CB, Salamone J, et al. Brain mechanisms underlying apathy. Journal of Neurology, Neurosurgery & Psychiatry 2019;90:302-312.

Apathy- Proposed mechanism

- Complex network of reciprocally connected cortical and subcortical brain regions, under the influence of the mesolimbic dopaminergic system.
- Internal valuation system must determine the subjective value of ongoing events in terms of hedonic or aversive potential + potential costs, including energy expenditure. Ventromedial prefrontal cortex (vmPFC)
- Mediating system integrates this reinforcer/cost information to activate the motor system towards particular goals. Ventral striatum (VS), anterior cingulate cortex (ACC) and mesolimbic dopamine (originating in VTA) form the mediating system.
- Motor system produces behaviour towards motivationally relevant stimuli. Hierarchically organised from complex/high-level to subcomponent/low-level and from goal-directed/cortical to habitual/sub-cortical. Posterior mid-cingulate cortex (pMCC), supplementary motor area (SMA), ACC and dorsal striatum (including the caudate and the putamen)
- Implication: measures to increase the incentivising value of rewards are an important therapeutic target



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Apathy- frequency and correlates

- Approx half of patients at some stage in the post-severe TBI period
- Baseline apathy was not associated with apathy 1 year later, suggesting reversibility
- Negative impact on rehabilitation efforts and social integration
- Anger, apathy, and dependency cause the greatest distress for caregivers

Rao V, McCann U, Bergey A, et al: Correlates of apathy during the first year after traumatic brain injury. Psychosomatics 54(4):403–404, 2013
Arnould A, Rochat L, Azouvi P, et al: A multidimensional approach to apathy after traumatic brain injury. Neuropsychol Rev 23(3):210–233, 2013
Marsh NV, Kersel DA, Havill JH, et al: Caregiver burden at 1 year following severe traumatic brain injury. Brain Inj 12(12):1045–1059, 1998

Apathy- management

- Avoid stressful situations and interference, clear and precise instructions, verbal reminders with cues, writing important information and encouraging patients to do this, persisting and helping the patient to persist in the use of tools and strategies
- Planned care itineraries and occupational and social activities, as well as cognitive-behavioural and family therapy

Wiart L, Luauté J, Stefan A, et al: Non pharmacological treatments for psychological and behavioural disorders following traumatic brain injury (TBI). A systematic literature review and expert opinion leading to recommendations. Ann Phys Rehabil Med 59(1):31–41, 2016

Apathy- management

- Studies consisted of single patients/small samples, with proper, adequately powered RCTs needed
- Available evidence suggests that psychostimulant medication may reduce apathy in some post-TBI (methylphenidate)
- 2nd line- cholinesterase inhibitors (donepezil)
- Dopamine agonists- amantadine

Plantier D, Luauté J, SOFMER group: Drugs for behavior disorders after traumatic brain injury: systematic review and expert consensus leading to French recommendations for good practice. Ann Phys Rehabil Med 59(1):42–57, 2016

Case 1 update

- Recently commenced on methylphenidate, improvement in concentration and memory but not in motivation
- Monitoring for any increase in aggression, anxiety, instability of mood or deterioration in sleep
- Ongoing

Case 2

- 66 year old married father, living with second wife overseas, project planner in Australia for work, ex-navy, lost control of vehicle at speed. Severe TBI (PTA 19 days) with DAI and subsequent stroke → R. hemiparesis
- Slow tempo slurred speech, erratic sleep, inconsistent engagement, rigidity, lability
  - SW: support wife to extends visa, work with ex-wife to sell jointly owned property, CTP, solicitor liaison
  - Neuropsych: capacity, recommendations to manage inflexibility and anxiety- validate, reassurance, time-out
  - Speech: cognitive-communication impairment- overinclusive, verbose, abrupt. “Stop-think-speak”, carer ed
  - OT: adaptive feeding equipment, shower chair, toilet frame- declined use on leave. Wheelchair-declined
  - Physio: mobility, lower-limb strengthening, hydrotherapy
  - Dietician: overweight, education
  - Case management: Lifetime care

Case 2

- Referred whilst an inpatient at rehab for management of anxiety and difficulty sleeping
- Excessive generalised worry, irritable, sleep difficulty (believed d/t environmental factors)
- Commenced on mirtazapine, helped with sleep, weight monitored
- Continued to be very rigid, controlling and emotional. Easily crying at a range of triggers frustration of discomfort of mattress, joy at improvements in physical function, overwhelmed at shopping centre

Emotional dyscontrol

- Unpredictable and rapidly changing emotions that are excessively intense relative to stimulus and not amenable to full voluntary control
- Prototypical form- pathological laughing and crying (PLC)/pseudobulbar affect/emotional incontinence
  - Severe disturbance in moment-to-moment emotional expression/regulation (weather), rather than the sustained, excessive and pervasive disturbances of emotion characteristic of mood disorders (climate)
  - Brief, stereotyped, intense, uncontrollable episodes of laughing/crying triggered by sentimentally trivial or neutral stimuli
- Affective lability and irritability
  - Brief episodes of congruent emotional expression not discretely paroxysmal, of variable intensity, and partially amenable to voluntary control or interruption by external events
  - Characteristically involves crying or laughing, may entail anxiety/irritability as well

Lauterbach EC, Cummings JL, Kuppuswamy PS: Toward a more precise, clinically -informed pathophysiology of pathological laughing and crying. Neurosci Biobehav Rev 37(8):1893–1916, 2013

Arciniegas DB, Wortzel HS: Emotional and behavioral dyscontrol after traumatic brain injury. Psychiatr Clin North Am 37(1):31–53, 2014

Emotional dyscontrol- frequency and correlates

- PLC: pathological crying is more than four times more common than pathological laughing, with the overall frequency of both types combined being approximately 16%, declines over the first year postinjury
- Estimates of prevalence of lability range from 33%–46% in the early postinjury period to 14%–62% in the late postinjury period

Roy D, McCann U, Han D, et al: Pathological laughter and crying and psychiatric comorbidity after traumatic brain injury. J Neuropsychiatry Clin Neurosci 27(4):299–303, 2015

Differentiating emotional dyscontrol

- PLC- ictal laughing (gelastic seizure) and ictal crying (dacrystic seizure)
- Lability- affective disorders, substance use disorders, idiopathic personality disorders

Emotional dyscontrol- management

- Counselling and education focussed on improving self-efficacy and self-regulation appear to effectively reduce affective lability and co-occurring behavioural dyscontrol
- Structured rehabilitation interventions focused on concurrently improving emotional regulation and cognitive performance

Cattelani R, Zettin M, Zoccolotti P: Rehabilitation treatments for adults with behavioral and psychosocial disorders following acquired brain injury: a systematic review. Neuropsychol Rev 20(1):52–85, 2010

Emotional dyscontrol- management

- Serotonergically and/or noradrenergically active antidepressants are effective treatments
- Selective serotonin reuptake inhibitors (SSRIs) are 1st line (sertraline, citalopram, and escitalopram)
- 2nd line- Methylphenidate (especially with slow processing speed or inattention), lamotrigine (especially with comorbid epilepsy), levodopa or amantadine, and anticonvulsants such as valproate or carbamazepine (especially with comorbid irritability/anger and aggressive/self-destructive behaviours)

Wortzel HS, Oster TJ, Anderson CA, et al: Pathological laughing and crying: epidemiology, pathophysiology and treatment. CNS Drugs 22(7):531–545, 2008

Arciniegas DB, Wortzel HS: Emotional and behavioral dyscontrol after traumatic brain injury. Psychiatr Clin North Am 37(1):31–53, 2014

Case 2 update

- Wife felt rigidity and controlling behaviour were not dissimilar to baseline
- Psychoeducation provided re biopsychosocial treatment options for lability- wife actually liked the increased emotional expression and both felt able to manage with behavioural interventions


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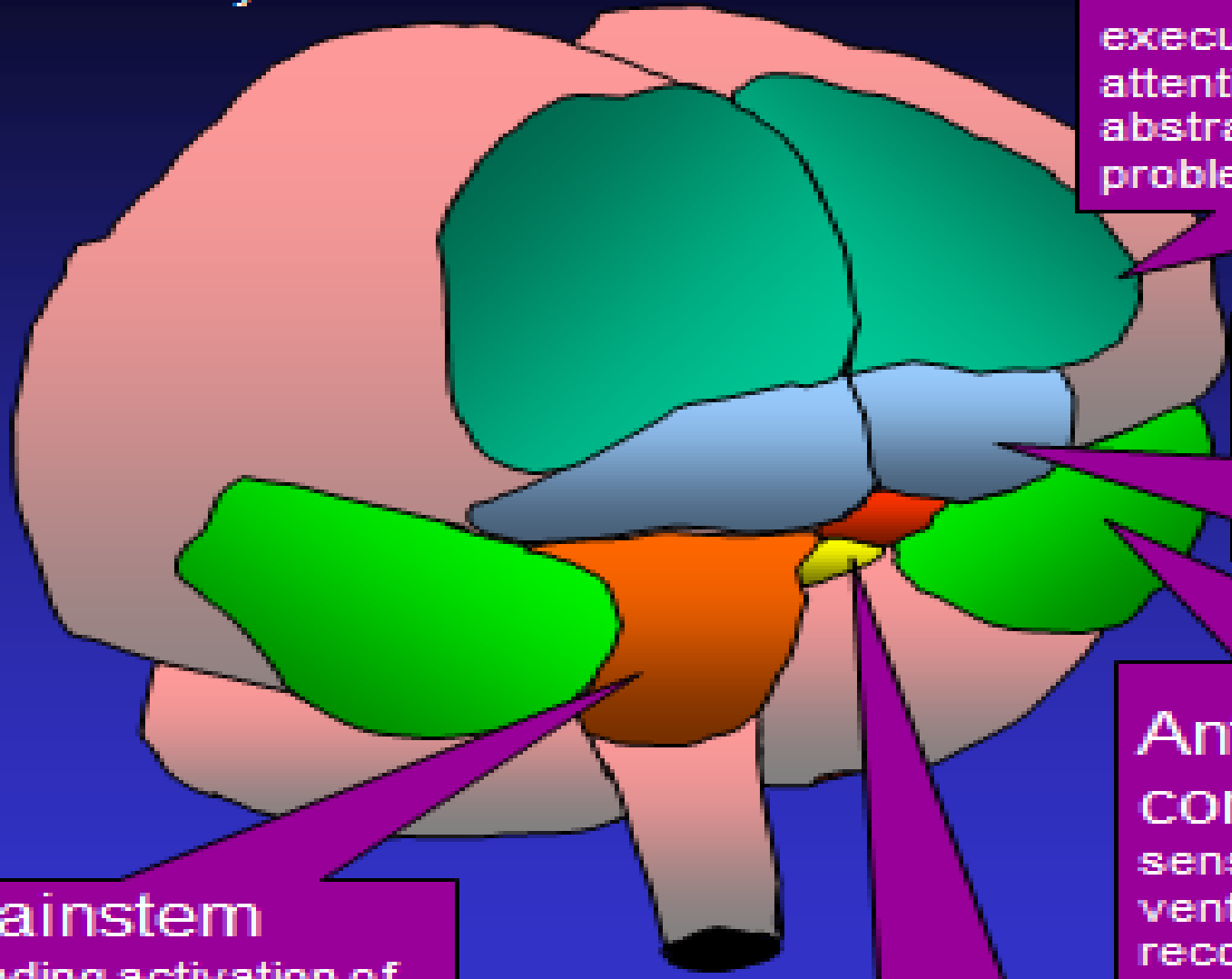
Severe behavioural disturbances in brain injury

- Or, what to do if the Positive Behaviour Support model isn't working for extreme behavioural disturbance.
  - **Aggression and severe compulsive behaviours**
  - **Sexually inappropriate behaviour**

Adapted from Arciniegas and Beresford
Neuropsychiatry: an introductory approach
Cambridge University Press 2001



Dorsolateral prefrontal cortex

executive function, sustained attention, memory retrieval, abstraction, judgement, insight, problem solving

Orbitofrontal cortex

emotional and social behaviour

Anterior temporal cortex

sensory-limbic interactions
ventral part involved in face recognition

Ventral brainstem

arousal, ascending activation of diencephalic, subcortical, cortical structures

Hippocampus

declarative memory

Why **not** to use medications

- Not good for our participants!
- Hard on their sensitive recovering brains, likely to interfere with neurorehabilitation, increase falls risk, have tolerability problems (weight gain, impaired mobility, sedation).
- So preface everything I say with first, strenuous attempts at behavioural and environmental management per PBS, with family support wherever possible.

When to use medications

- In an adequately resourced setting, when all feasible behavioural and environmental interventions have been tried and the behaviour is causing acute distress or danger to the participant, other participants, carers, family or others.

- In an inadequately resourced setting, do whatever you have to do to keep the participant and everyone else (including you) safe from harm, then when the situation has resolved demand extra resources long and loud!

Pharmacotherapy of aggression

- Again, medication is a last resort when all other options have been tried
- How do we assess the evidence?
- Not many studies, quality poor - fair

Mehta, Swati, et al. "Pharmacological management of agitation among individuals with moderate to severe acquired brain injury: a systematic review." Brain Injury 32.3 (2018): 287-296.

Williamson, David, et al. "Pharmacological interventions for agitated behaviours in patients with traumatic brain injury: a systematic review." BMJ open 9.7 (2019): e029604.

Table 2. Modified Sackett's levels of evidence (14).

Level	Research Design	Description
Level 1a	RCT	More than 1 RCT with PEDro score ≥6 Includes within subjects comparison with randomized conditions and crossover designs
Level 1b	RCT	1 RCT with PEDro ≥ 6
Level 2	RCT	RCT, PEDro < 6
	Prospective controlled trial	Prospective controlled trial (not randomized)
	Cohort	Prospective longitudinal study using at least two similar groups with one exposed to a particular condition
Level 3	Case–control	A retrospective study comparing conditions including historical controls
Level 4	Pre–post test	A prospective trial with a baseline measure, intervention, and a post-test using a single group of subjects
	Post-test	A prospective intervention study using a post intervention measure only (no pre-test or baseline measurement) with one or more groups
	Case series	A retrospective study usually collecting variables from a chart review
Level 5	Observational study	Using cross-sectional analysis to interpret relations
	Clinical consensus	Expert opinion without explicit critical appraisal, or based on physiology, biomechanics or 'first principles'
	Case reports	Pre–post or case series involving one subject

PEDro = Physiotherapy Evidence Database.

Evidence for **anticonvulsants**

Level 4 evidence that **carbamazepine, valproate and lamotrigine** may reduce agitation after acquired brain injury.

Evidence for **antidepressants**

Level 4 evidence that **amitriptyline** may improve agitation post brain injury.

Level 4 evidence that **sertraline** may be effective in reducing agitation among participants who have had an ABI for greater than 3 months**.**

Level 1b evidence that sertraline may NOT improve agitation in individuals within 2 weeks of ABI.

Evidence for **amantadine**

Level 1a evidence that **amantadine** effectively reduces symptoms of agitation among those with acquired brain injury.

Evidence for **antipsychotics**

Level 3 evidence that haloperidol may NOT be effective in managing agitation behaviour among individuals with ABI.

Level 4 evidence that **droperidol, quetiapine,** and **ziprasidone** are effective in reducing agitation post-brain injury.

Evidence for **lithium**

Level 4 evidence that **lithium** is effective in reducing agitation post-brain injury.

Evidence for **beta-blockers**

Level 1b evidence that **pindolol** is effective in reducing episodes of agitation among individuals with acquired brain injury.

Level 1b evidence that **propranolol** may not be effective in reducing agitation episodes, but may be effective at reducing the intensity of agitation among individuals with ABI.

Evidence summarised I

Strong evidence for **amantadine.**

Weak evidence for **valproate, carbamazepine, lamotrigine, amitriptyline, sertraline** (after three months, not before two weeks)**, lithium, droperidol, quetiapine, ziprasidone, pindolol,** and **propranolol** (for reducing intensity of agitation only).

Mehta, Swati, et al. "Pharmacological management of agitation among individuals with moderate to severe acquired brain injury: A systematic review." Brain injury 32.3 (2018): 287-296.

Evidence summarised II

Propranolol, methylphenidate, valproic acid and **olanzapine** were the only agents suggesting a potential benefit in reducing agitation, anger or irritability. **Amantadine** showed mixed results.

Williamson, David, et al. "Pharmacological interventions for agitated behaviours in patients with traumatic brain injury: a systematic review." BMJ open 9.7 (2019): e029604.

What I do

Olanzapine (be careful if overweight or diabetic) 2.5 – 20mg/day

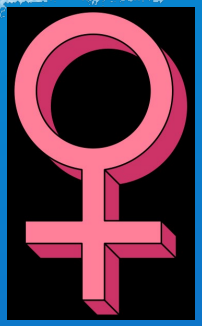
+/- valproate (NB unsafe in pregnancy) 200 – 3000mg/day

+/- propranolol 80 – 480mg/day (only higher doses in acute medical settings).

Occasionally add amantadine. Haven't used methylphenidate but one intriguing older study suggests perhaps we should!

Mooney, George F., and Leonard J. Haas. "Effect of methylphenidate on brain injury-related anger." Archives of physical medicine and rehabilitation 74.2 (1993): 153-160.

Sexually intrusive behaviour



- This can be dismissed as “Benny Hill”-type behaviour but in fact can be incredibly distressing to others, especially those with a sexual trauma history (at least 25% of women and 10% of men, likely higher in care workers)

Types of sexually intrusive behaviour

- Sexual talk
- Non-genital touching
- Self-exposure
- Public masturbation
- Genital touching
- Sexual coercion
- Sexual assault

How common is it?

- Very. In a survey of rehabilitation professionals in a brain injury unit, 70% of respondents reported that sexual touching was a common problem at their facility, and 20% reported that the use of sexual force by patients was common.

Bezeau, Scott C., Nicholas M. Bogod, and Catherine A. Mateer. "Sexually intrusive behaviour following brain injury: Approaches to assessment and rehabilitation." Brain Injury 18.3 (2004): 299-313.

Who develops sexually intrusive behaviour?

- Pre-injury factors
  - Antisocial personality
  - History of sexually inappropriate behaviour
- Injury factors
  - Hypersexuality more common after medial basal-frontal, diencephalic injury.
  - Much more rarely Kluver-Bucy syndrome after bilateral temporal lesions
- Environment factors
  - Ward layout, routines, crowding

Pharmacological treatments

Thorough medical workup (history, examination, investigations) to exclude other causes of hypersexuality, especially complex partial seizure activity. If all clear, three options:

1. **SSRIs eg Lovan, Zoloft, Lexapro, Aropax etc**
2. **Anti-androgens**
3. **LHRH agonists**

Plantier, D., and J. Luauté. "Drugs for behavior disorders after traumatic brain injury: systematic review and expert consensus leading to French recommendations for good practice." Annals of physical and rehabilitation medicine 59.1 (2016): 42-57.

SSRIs

- Good first line agents
- Generally well-tolerated (watch for nausea, restlessness, sleep disturbance)
- Act by increasing serotonin levels in pudendal nerves: leads to reduced sexual desire as well as problems with erection and ejaculation
- Which one? **Paroxetine** and **fluvoxamine** have the highest rates of sexual side effects but best off using the drug team doctor is most comfortable prescribing

Anti-androgens

- If SSRIs are ineffective or poorly tolerated, a number of drugs have anti-testosterone effects, including:
  - **Cyproterone acetate (Androcur)**
  - **Spironolactone (Aldactone, diuretic)**
  - **Cimetidine (Tagamet, anti-ulcer)**
  - **Ketoconazole (antifungal)**
  - **Risperidone**

LHRH agonist

- Depo-Provera (medroxyprogesterone acetate)
- A study on 40 men (not all had TBI) treated with medroxyprogesterone after sexual aggression showed a recurrence rate of 18% vs. 58% without treatment
- Generally said to be well tolerated but limited literature available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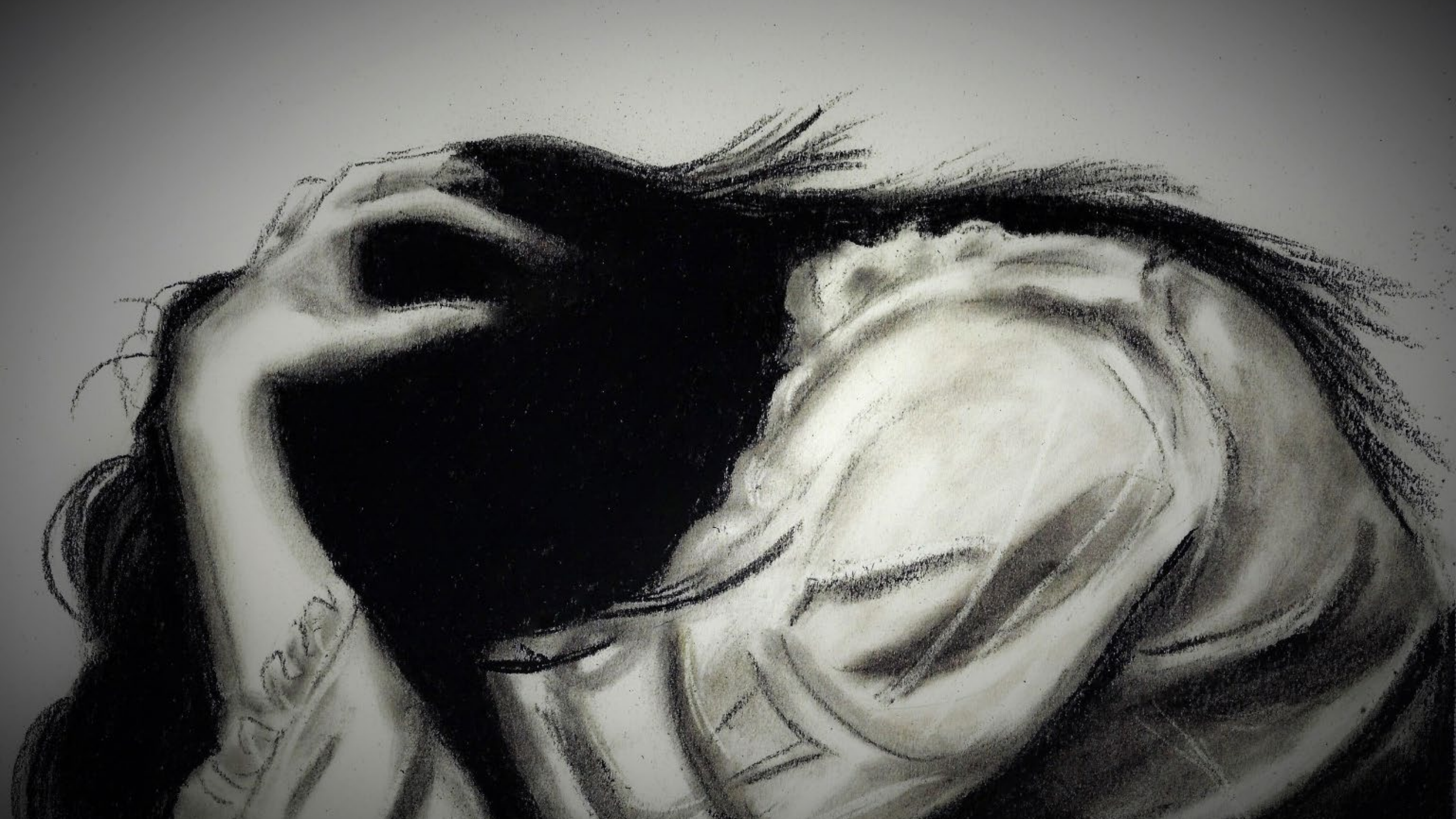
Anxiety and depression in TBI



“Fran”: anxiety and depression post TBI

- Fran is a 30 year old agricultural scientist, severely injured in a head in northwestern NSW 5 years ago. She lives with her partner Steve.
- She sustained a severe brain injury, perforated ileum, fractured C5 and C6 vertebrae as well as maxillofacial injuries and a fractured fibula. She spent four weeks at a large regional hospital and underwent burrhole surgery, then a further 10 weeks at a Brain Injury Unit. She continues to have a right homonymous hemianopia.
- After a long period of emotional numbness she is now beginning to feel things more often and particularly anxiety which can be quite paralysing at times. She has had experiences of waking up terrified on some days while other days are less concerning. She also suffers from initial insomnia, low energy, anhedonia and low mood. Recently her mood had become so low that she had begun actively considering a means of suicide and contacted Lifeline.

“Fran”: initial assessment continued

- She has a premorbid history of anxiety and depression and in fact made a suicide attempt of sorts at the age of 19 when she lay on some train tracks for a period of time and then later had contact with Lifeline.
- There is no other significant medical history. She has no allergies, is a non-smoker, rarely drinks alcohol and does not use drugs.
- She was raised in Sydney, the youngest of four children. Her father was a scientist and her mother is a secretary in an accounting firm. There were no developmental difficulties and she denies any traumas or stresses in childhood. She describes her childhood as pleasant and relaxed. She thinks he was a quiet and bookish child whose reserved manner caused difficulties at school, particularly in high school where she was in what she called the ‘outcasts group’ and experienced significant bullying. She was sufficiently affected by this to have a much poorer HSC result than she was expecting. She repeated her HSC at TAFE and studied science at university with honours, going on to complete a PhD about a year before her injury.

“Fran”: initial assessment continued

- She was on an SSRI antidepressant for about a year in 2009 when she developed some bothersome checking behaviour involving locks and doors to the point where she was having difficulty leaving the house. These mild OCD and depressive symptoms improved but after a year she stopped the escitalopram.
- Currently Fran is continuing to struggle with low mood. She said ‘I feel pointless’ and is frustrated that her brain is working less effectively than previously. She had difficulties giving an academic lecture recently and answering questions after it.
- She is seeing a clinical psychologist with extensive experience in brain injury approximately every three weeks.
- On mental state examination Fran was a slim woman with glasses who had a mildly anxious but cooperative manner. She required a little bit of support and redirection by Steve during the interview but generally did well. She scored 40 on a Centre for Epidemiologic Studies Depression Scale, consistent with a very significant depression and moderately on scales that measured anxiety and OCD symptoms.

“Fran”: initial assessment continued

- When first seen Fran was a young woman who has had a severe traumatic brain injury 18 months ago occurring against a background of high trait anxiety, OCD and recurrent depression. She now has symptoms of a major depressive disorder as well as generalised anxiety and intermittent OCD symptoms.
- My recommendations were that Fran continued to work closely with her psychologist and recommence escitalopram. I have spoken to Fran and Steve about how to manage escalating suicidality or severe low mood including how to contact the mental health extended hours team in a crisis.

“Fran”: subsequent developments

- Year one:
  - Initial improvement with SSRI and CBT. Declined online anxiety management module I had recommended.
  - Development of panic attacks up to 8x/week, SSRI increased in dose, improved
- Year two:
  - Psychologist sessions started to wind up
  - Fran began to describe feelings of numbness, detachment
  - “She tends to have a panic response to ordinary negative events during the day, followed by self blame and irrational thoughts”
  - Sitcom theme song began playing repeatedly in her head
  - Further engagement with psychologist recommended

.

“Fran”: subsequent developments

- Year three:
  - Returned after five month gap markedly deteriorated. No obvious cause. Markedly depressed with feelings of despair, pointlessness.
  - SSRI increased further, re-referred to clinical psychologist
  - Improvement when reviewed next
  - Recurrent feelings of being overwhelmed, occasional panic attacks still.
  - Unexpected pregnancy, not recognised until week 14! Antenatal care hastily arranged.
  - Partner also experiencing anxiety, referred.
  - Seen by mental health team perinatally and by psychologist after baby’s birth.
- Year four:
  - Mood swings more severe. Feelings of hopelessness, thoughts of suicide, quite intense.
  - Emergence of postnatal depression, SSRI changed, olanzapine added.
  - Mood swings more extreme, more demands on psychologist.
  - Thoughts of self harm developing. Partner not sure he can continue to manage.
  - Currently on holiday: work in progress.

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“Fran”: current thinking

- Brain injury very important factor, but there is more
- Postnatal depression
- Are anxiety symptoms just intensification of premorbid problems
- Emergence of trauma/personality symptoms (mood swigs, suicidality, threats against partner)
- Relationship issues
- Medication revised
- Regular liaison with clinical psychologist, OT, CM
- Consideration of MH admission or DBT group

Antidepressants in TBI

- Widely used but some recent cautions.
- Two studies suggest that antidepressants may not improve depressive symptoms in TBI, and may actually worsen cognition.
- This doesn't really tally with clinical experience so more studies are needed.
- Other studies suggest they are beneficial and may help with cognition.
- Sertraline and es/citalopram most commonly used.

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Kreitzer, Natalie, et al. "The effect of antidepressants on depression after traumatic brain injury: a meta-analysis." *The Journal of head trauma rehabilitation* 34.3 (2019): E47-E54.

Failla, Michelle D., et al. "Effects of depression and antidepressant use on cognitive deficits and functional cognition following severe traumatic brain injury." *The Journal of head trauma rehabilitation* 31.6 (2016): E62.

Psychosis in TBI


ψυχική

“James”: brain injury and psychosis

- James sustained a severe traumatic brain injury in a four metre cliff fall with a GCS at the scene of 3-6/15 and a duration of PTA of three weeks. CT brain findings showed bilateral punctate intra axial haemorrhage and a small right posterior parietal haemorrhage consistent with a possible subdural. He required urgent insertion of an extraventricular drain and spent eight days in ICU at a major Sydney hospital with a further week in a high dependency unit before being transferred to the Brain Injury Unit and being discharged on after five weeks. His admission was complicated by right basilic vein and large right ilio-femoral DVTs which required heparinisation and warfarinisation.
- While recovering well cognitively, James developed psychotic symptoms about three weeks after his injury including persecutory delusions and the development of delusions of misidentification (Capgras delusions) regarding his parents, believing that they had been substituted by others. This sense of unreality subsequently extended to his home to a belief that perhaps he was not alive and had been transferred to some other plane of existence. He described this as “a glitch in the matrix”.

“James”: initial assessment continued

- A trial of olanzapine up to 7.5mg daily was associated with sedation and sleeping for more than 12 hours daily but did not appear to significantly ameliorate the symptoms. James cross tapered over to risperidone and when I first saw him post-discharge was taking 2.5mg of olanzapine and 2mg twice daily of risperidone. He had noticed the emergence of sexual side effects on the risperidone but was not unduly distressed by this at that stage.
- Apart from these very unsettling symptoms which at times have been so distressing for James that he has had suicidal thoughts and has asked his parents to kill him the rest of his rehabilitation has progressed well. He has not had aggressive or violent thoughts about his parents. Other psychosis symptoms that have been present have included thought broadcasting and what sounds like some grandiose delusions at one point in his recovery where he thought he might be some kind of god, but these have both resolved.
- James was referred to a psychologist for some depressive symptoms and low self-esteem in 2015 but his mood improved after he got a job working in a restaurant and he did not end up seeing the psychologist. He has significant history of substance abuse including binge alcohol use, regular daily cannabis, and periodic use of LSD. His parents also noted that he had an interest in fairly abstract philosophy and science fiction prior to his injury.
- His medical history is significant for a depressed parietal skull fracture at the age of four. He also had low grade asthma as a child and was quite allergic to pollen. There is no other significant medical history. He was a smoker until the time of his injury but he is not currently smoking.
- There is a family history of psychosis with a paternal great aunt who developed schizophrenia.

“James”: initial assessment continued

- On mental state examination James was a slim well looking young man who was polite and attentive throughout the interview. He answered questions freely and was receptive to discussion about the pathological nature of these experiences as a result of his traumatic brain injury. He showed no signs of motor restlessness or any other extrapyramidal signs.
- In summary James described the emergence of an organic psychotic disorder following a traumatic brain injury with the development of Capgras syndrome features of delusional misidentification. At times this feeling had been extended into a general sense of delusional mood or unreality but there was a sense that these symptoms had improved a little particularly with the recent change from olanzapine to risperidone.
- I asked James to stop the olanzapine and continue on risperidone 2mg twice daily. We discussed potential motor and hormonal adverse effects.
- I recommended regular cognitive challenge with a clinical psychologist familiar with psychosis.

“James”: subsequent progress

- Motor restlessness on risperidone so dose reduced to 2mg.
- Generally ok but some odd preoccupations and mood variability. Parents thought “close to normal”.
- Elevated prolactin, sexual side effects, risperidone reduced to 1mg.
- More prominent irritability. *We discussed three possible explanations for his increasing irritability: 1. That this is just a natural reaction to the limitations that have been placed on him and the frustrations of being in the care of his parents at present. 2. That he has some ongoing irritability and affective liability from his head injury which had previously been masked by the higher dose of risperidone. 3. That this could represent an “at risk state” for a recurrence of his psychosis.*
- SSRI added (escitalopram)

James: subsequent progress

- Referred to early psychosis (EP)service for co-management
- Risperidone increased to 2mg again because of lability, “strange” thoughts.
- Persistent symptoms even after risperidone increased to 2.5mg, EP team changed him to aripiprazole. Sexual function improved, but more preoccupied with whether experiences are real, whether parents are lying to him.
- EP team changed him to quetiapine, persistent symptoms.
- I began to lobby for a trial of clozapine, resisted by family and EP team less enthusiastic.
- Remained on clozapine. Collapse on skiing trip, ?? seizure, ? functional.
- Aripiprazole added

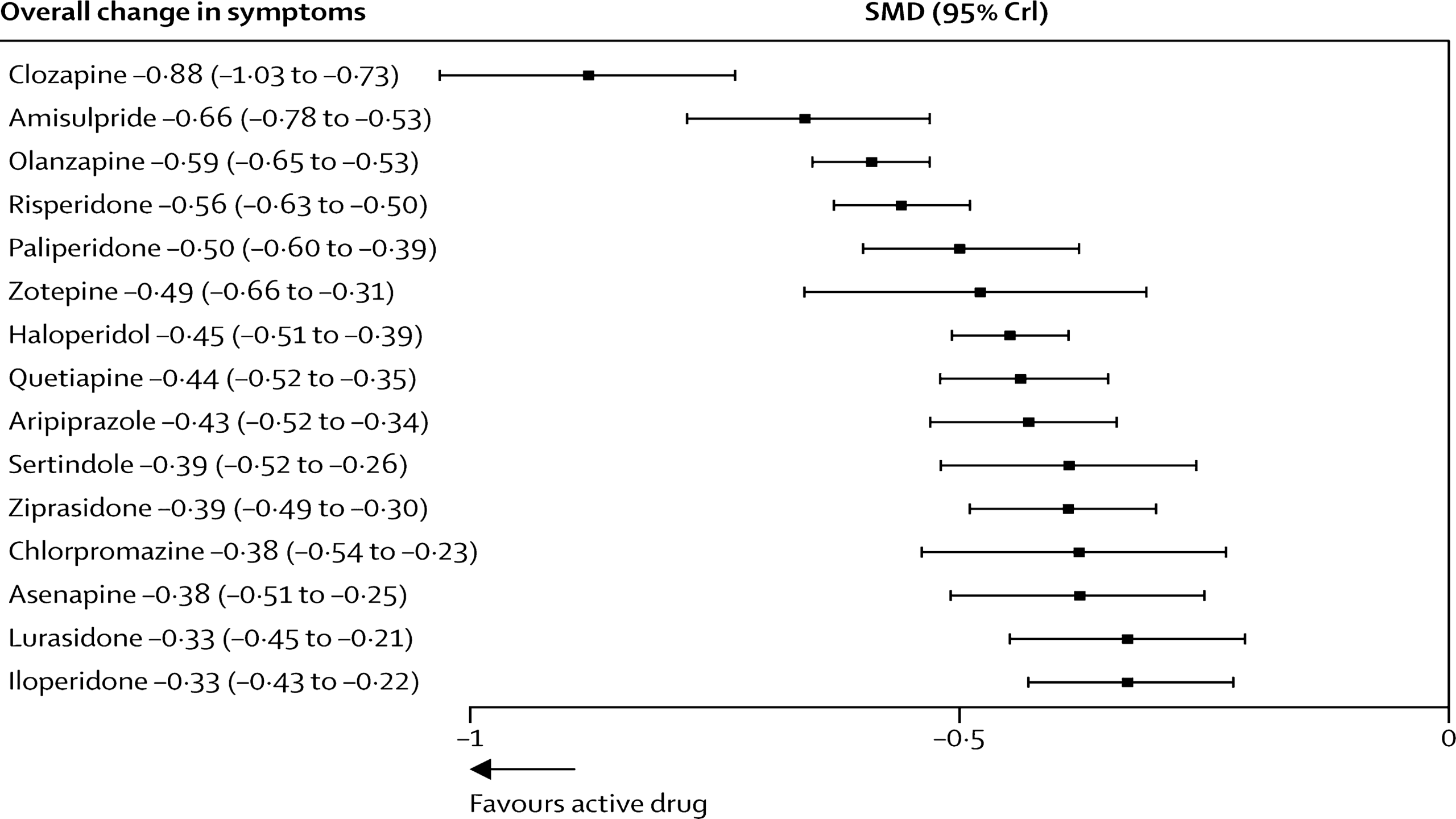
“James”: subsequent progress

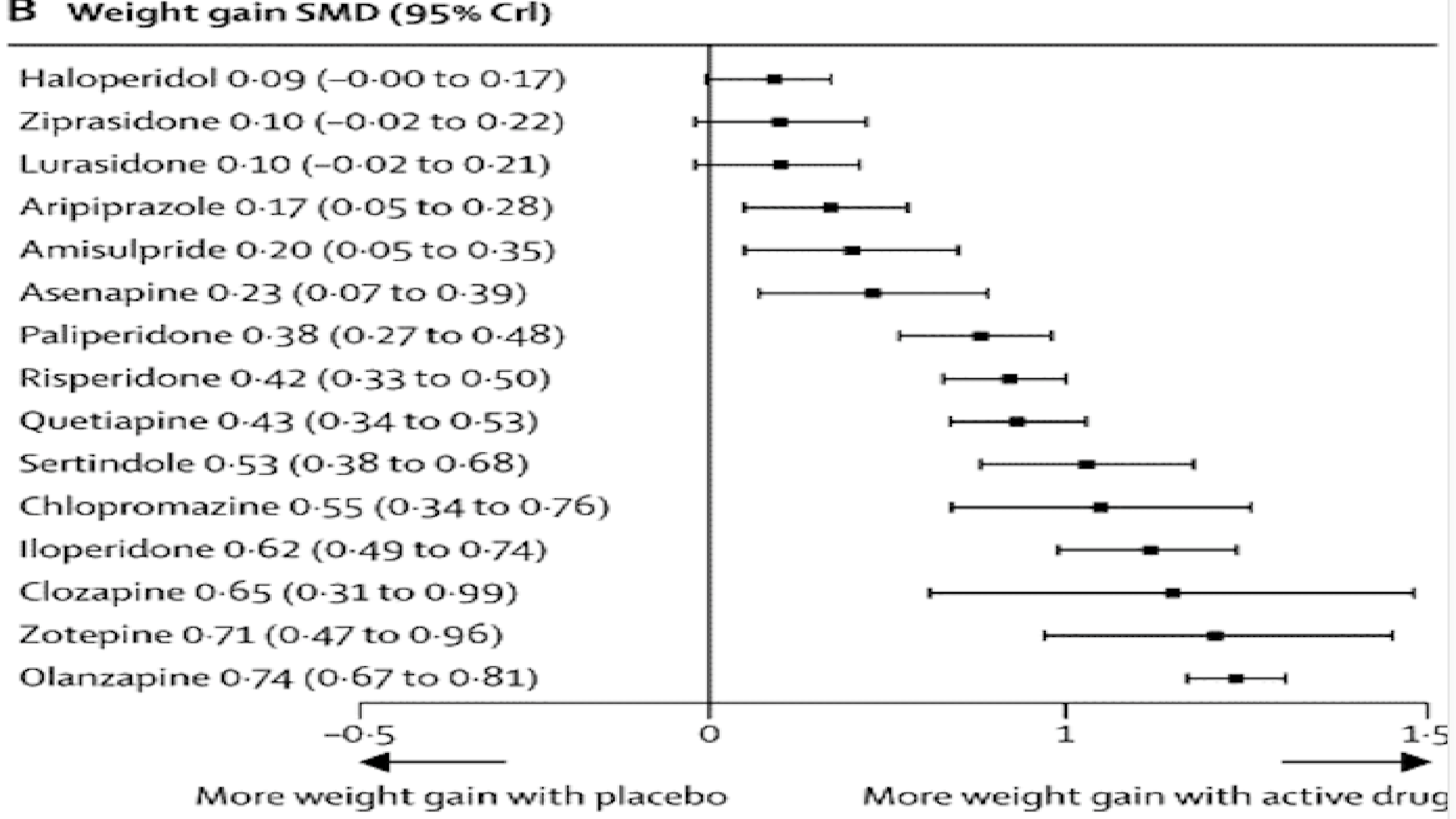
- Persistent disability, doing some bar work. DSP application made.
- After two years, moved with family to regional area, care transferred.
- My last entry: *James remains on quetiapine 300 mg at night and aripiprazole 5 mg in the morning. He is generally doing okay, but his mood remains quite up and down. He starts the day with feelings of motivation and positivity which rapidly dissipate and by the early afternoon he says "I feel like giving up, everything is too difficult". He is also bothered by apparent blepharospasm of the left eye which seems to worsen at times of stress. At a recent family wedding in southwestern Sydney James became very tired and had some quite pronounced dissociative symptoms with concerns about the nature of reality and whether he existed which were very troubling for him. He was able to be reassured by his father and the symptoms had settled by the next morning but he was keen to discuss their significance with his parents the next evening.*
- *In summary, James seems to be doing well but he does have some residual psychosis symptoms I think, including some positive symptoms and significant negative and cognitive symptoms. I have always favoured James having a trial of clozapine but he and his family have been cautious about this. It may be something that can be revisited by Dr Xxxxx as it would be good to see whether something can be done to make his recovery more complete.*

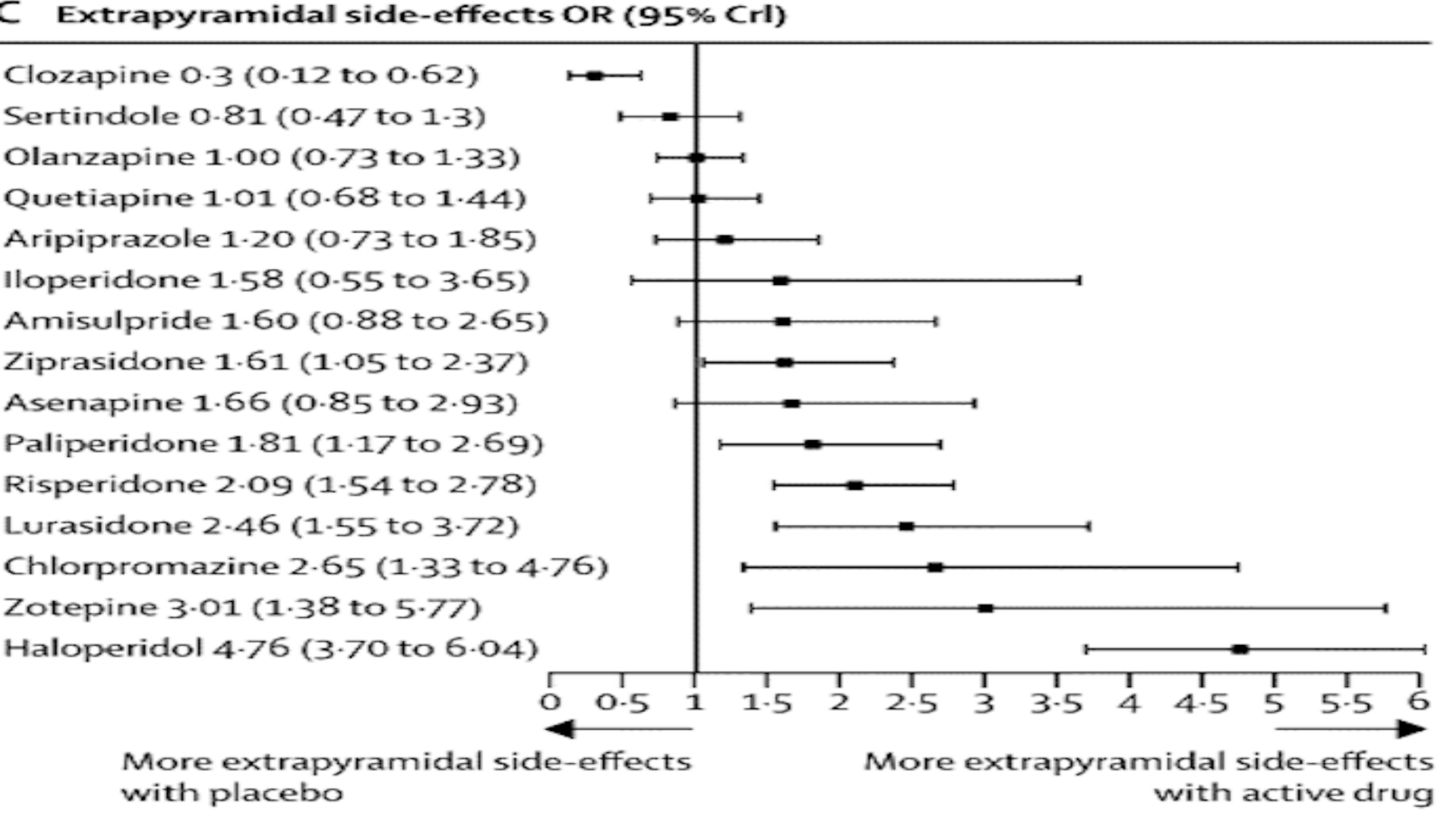
Psychosis and brain injury

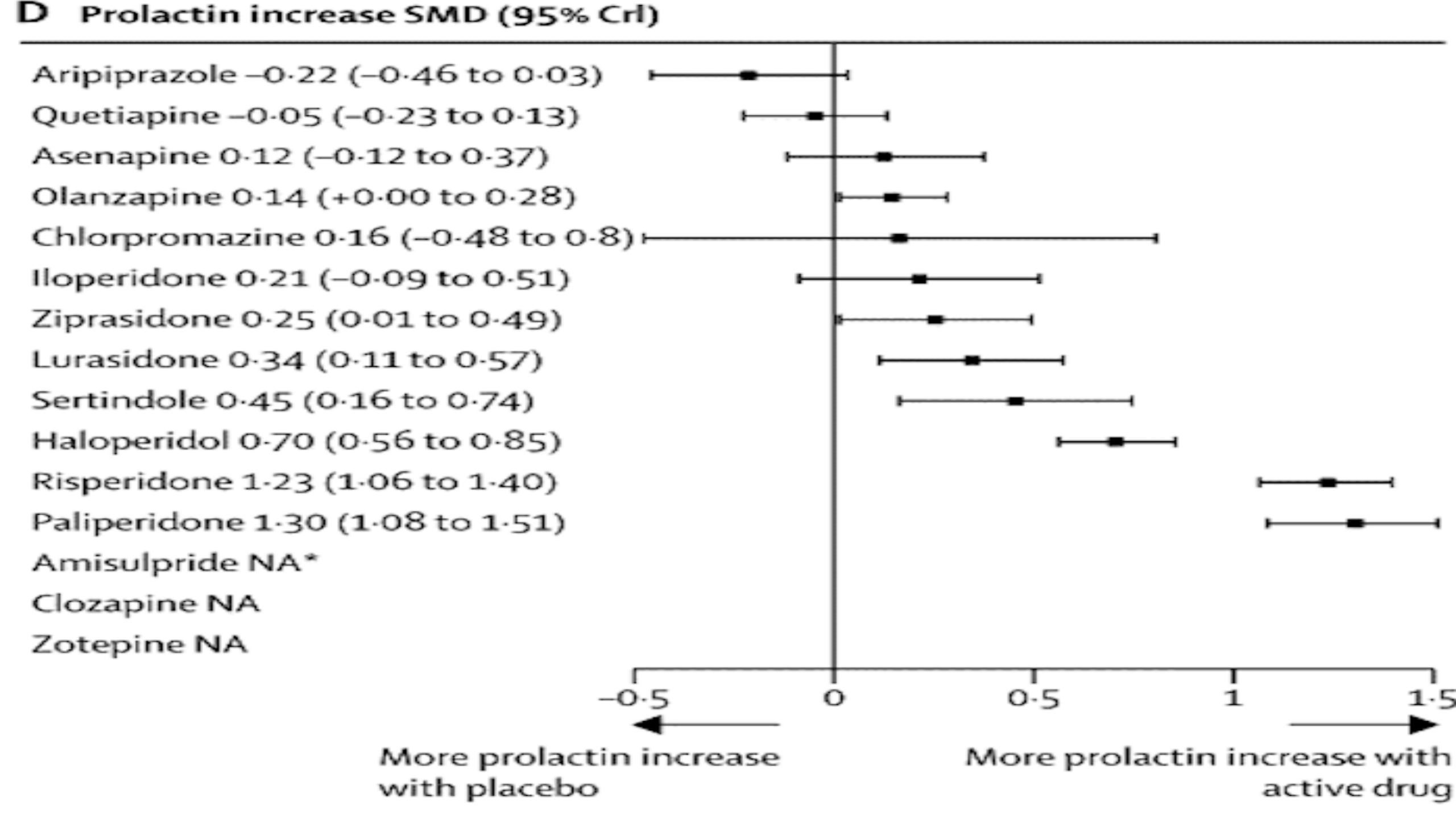
- Psychosis in TBI is rare, but there is a 2 to 3 fold increase in the risk of psychosis in head injured individuals compared to the general population (8% cf 2%)
- More sensitive to antipsychosis side effects, especially sexual and motor effects (restlessness, parkinsonism, dystonia).
- So, which to choose?

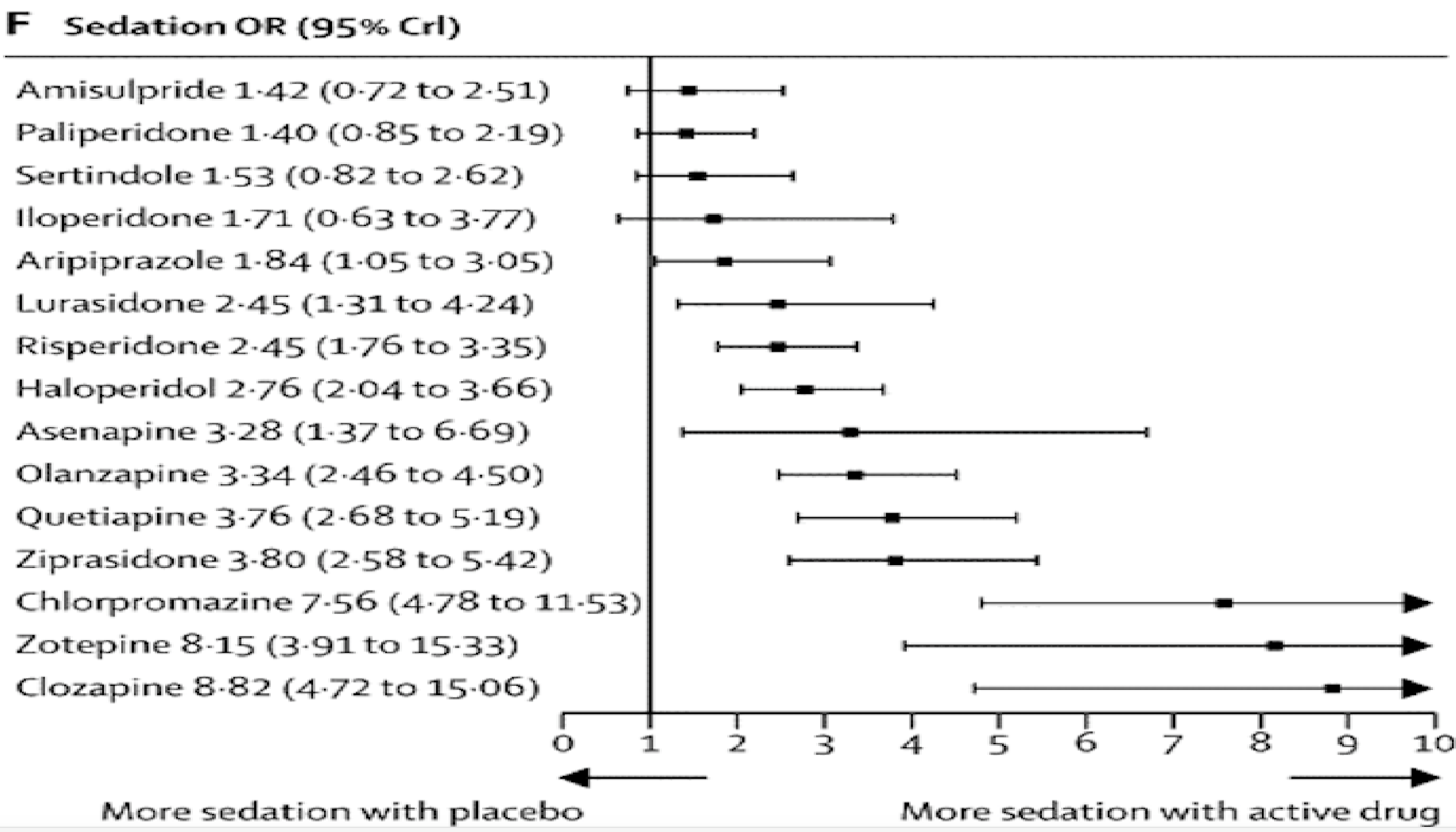
Leucht, Stefan, et al. "Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis." *The Lancet* 382.9896 (2013): 951-962.


Overall change in symptoms
SMD (95% CrI)
Clozapine –0·88 (–1·03 to –0·73)
Amisulpride –0·66 (–0·78 to –0·53)
Olanzapine –0·59 (–0·65 to –0·53)
Risperidone –0·56 (–0·63 to –0·50)
Paliperidone –0·50 (–0·60 to –0·39)
Zotepine –0·49 (–0·66 to –0·31)
Haloperidol –0·45 (–0·51 to –0·39)
Quetiapine –0·44 (–0·52 to –0·35)
Aripiprazole –0·43 (–0·52 to –0·34)
Sertindole –0·39 (–0·52 to –0·26)
Ziprasidone –0·39 (–0·49 to –0·30)
Chlorpromazine –0·38 (–0·54 to –0·23)
Asenapine –0·38 (–0·51 to –0·25)
Lurasidone –0·33 (–0·45 to –0·21)
Iloperidone –0·33 (–0·43 to –0·22)
–1
–0·5
0
Favours active drug


B Weight gain SMD (95% CrI)
Haloperidol 0·09 (–0·00 to 0·17)
Ziprasidone 0·10 (–0·02 to 0·22)
Lurasidone 0·10 (–0·02 to 0·21)
Aripiprazole 0·17 (0·05 to 0·28)
Amisulpride 0·20 (0·05 to 0·35)
Asenapine 0·23 (0·07 to 0·39)
Paliperidone 0·38 (0·27 to 0·48)
Risperidone 0·42 (0·33 to 0·50)
Quetiapine 0·43 (0·34 to 0·53)
Sertindole 0·53 (0·38 to 0·68)
Chlopromazine 0·55 (0·34 to 0·76)
Iloperidone 0·62 (0·49 to 0·74)
Clozapine 0·65 (0·31 to 0·99)
Zotepine 0·71 (0·47 to 0·96)
Olanzapine 0·74 (0·67 to 0·81)
–0·5
0
1
1·5
More weight gain with placebo
More weight gain with active drug


C Extrapyramidal side-effects OR (95% CrI)
Clozapine 0·3 (0·12 to 0·62)
Sertindole 0·81 (0·47 to 1·3)
Olanzapine 1·00 (0·73 to 1·33)
Quetiapine 1·01 (0·68 to 1·44)
Aripiprazole 1·20 (0·73 to 1·85)
Iloperidone 1·58 (0·55 to 3·65)
Amisulpride 1·60 (0·88 to 2·65)
Ziprasidone 1·61 (1·05 to 2·37)
Asenapine 1·66 (0·85 to 2·93)
Paliperidone 1·81 (1·17 to 2·69)
Risperidone 2·09 (1·54 to 2·78)
Lurasidone 2·46 (1·55 to 3·72)
Chlorpromazine 2·65 (1·33 to 4·76)
Zotepine 3·01 (1·38 to 5·77)
Haloperidol 4·76 (3·70 to 6·04)
0 0·5 1 1·5 2 2·5 3 3·5 4 4·5 5 5·5 6
More extrapyramidal side-effects with placebo
More extrapyramidal side-effects with active drug


D Prolactin increase SMD (95% CrI)
Aripiprazole –0·22 (–0·46 to 0·03)
Quetiapine –0·05 (–0·23 to 0·13)
Asenapine 0·12 (–0·12 to 0·37)
Olanzapine 0·14 (+0·00 to 0·28)
Chlorpromazine 0·16 (–0·48 to 0·8)
Iloperidone 0·21 (–0·09 to 0·51)
Ziprasidone 0·25 (0·01 to 0·49)
Lurasidone 0·34 (0·11 to 0·57)
Sertindole 0·45 (0·16 to 0·74)
Haloperidol 0·70 (0·56 to 0·85)
Risperidone 1·23 (1·06 to 1·40)
Paliperidone 1·30 (1·08 to 1·51)
Amisulpride NA*
Clozapine NA
Zotepine NA
–0·5
0
0·5
1
1·5
More prolactin increase with placebo
More prolactin increase with active drug


F Sedation OR (95% CrI)
Amisulpride 1·42 (0·72 to 2·51)
Paliperidone 1·40 (0·85 to 2·19)
Sertindole 1·53 (0·82 to 2·62)
Iloperidone 1·71 (0·63 to 3·77)
Aripiprazole 1·84 (1·05 to 3·05)
Lurasidone 2·45 (1·31 to 4·24)
Risperidone 2·45 (1·76 to 3·35)
Haloperidol 2·76 (2·04 to 3·66)
Asenapine 3·28 (1·37 to 6·69)
Olanzapine 3·34 (2·46 to 4·50)
Quetiapine 3·76 (2·68 to 5·19)
Ziprasidone 3·80 (2·58 to 5·42)
Chlorpromazine 7·56 (4·78 to 11·53)
Zotepine 8·15 (3·91 to 15·33)
Clozapine 8·82 (4·72 to 15·06)
0
1
2
3
4
5
6
7
8
9
10
More sedation with placebo
More sedation with active drug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OCD

- Lack of clarity about emergence of de novo obsessive-compulsive behaviour after TBI
- Although some outlier studies, most show similar low rate to gen population
- Clinically thought different in character, with emergence of compulsive or stereotyped behaviours post-TBI with minimal anxiety perhaps better classified as obsessive compulsive personality

Gould KR, Ponsford JL, Johnston L, et al: The nature, frequency and course of psychiatric disorders in the first year after traumatic brain injury: a prospective study. Psychol Med 41(10):2099–2109, 2011

OCD

- Obsessive thoughts, urges or images which the individual tries to suppress, often associated with fears about contamination, are far less common, or intrusive, than compulsive tendencies to maintain order
- Compulsive behaviour after TBI can be a coarsening of pre-accident personality characteristics, such that a person who was always methodical and organised exhibits a more concrete or rigid style of thinking leading to stereotyped behaviour patterns
- Working memory impairment with lack of confidence about whether or not an action (turning off the gas, electrics, etc.) has been carried out, can → checking rituals which then develop as a habit response

OCD

- Figee et al. reviewed 37 case reports of patients with acquired OCD due to acquired brain injury and suggest that lesions in the cortico-striato-thalamic circuit, parietal and temporal cortex, cerebellum and brainstem may induce compulsivity
- Hoarding behaviour/abnormal “collecting drives”, seems to be associated with an inability to decide what is useful and should be retained
- Post traumatic hoarding behaviours have been associated with mesial prefrontal damage

Figee M, Wielaard I, Mazaheri A, Denys D: Neurosurgical targets for compulsivity: what can we learn from acquired brain lesions? Neurosci Biobehav Rev 37: 328–339, 2013

Anderson SW, Damasio H, Damasio A: A neural basis for collecting behaviour in humans. Brain 128: 201–212, 2005

PTSD

- Encompasses emotional, behavioural and cognitive symptoms that arise following exposure to significant threat to life or serious physical injury
- Intrusion symptoms (nightmares, intrusive thoughts, dissociative reactions), behavioural and cognitive avoidance of trauma reminders, negative cognition or mood (anhedonia, negative expectations) and heightened arousal (sleep disturbance, impaired concentration)

PTSD

- Once believed alterations of consciousness with TBI precluded formation of a trauma memory
- Now recognised that PTSD can develop following TBI
  - unconscious encoding of affective and sensory experiences (sights and smells) associated with traumatic event
  - conscious encoding of some aspects of the event, reconstruction of the trauma memory from secondary sources and memory of circumstances surrounding the event that also may be psychologically traumatic (e.g. sights at the scene of accident after consciousness regained)

PTSD

- TBI may impede recovery from psychological trauma, and PTSD may impede recovery from TBI
- Mechanisms not fully understood, thought that the cumulative burden of each condition on neural and associated neurocognitive function may contribute to impediments in recovery

PTSD

- Guided by PTSD treatment in non-TBI population. CBT interventions, including those with exposure components, have been successfully implemented within a broad range of TBI severities
- General consensus in prescribing to patients with PTSD and a TBI history is to start with low dosages and titrate slowly, be cognizant of possible drug interactions, and use caution when potential medication side effects might increase risk of TBI-associated problems such as cognitive deficits, sensory and balance issues, and seizures

- Scott JC, Harb G, Brownlow JA, et al: Verbal memory functioning moderates psychotherapy treatment response for PTSD-Related nightmares. Behav Res Ther 91:24–32, 2017


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Case- background

- 56 year old single male, DSP, daily carers, living with his mother in their own home, working in telecommunications until made redundant 15 years ago
- 1st TBI- 2.5 years ago bike v car. PTA 27 days, R.sided subdural and frontal/parietal contusions, likely DAI. Acute admission 3 weeks, rehab admission 6 weeks. D/C physically independent, slowed speech and social communication deficits, main goal to return to exercising most of the day
- 2nd TBI- 6 months later ?secondary to seizure. PTA <1 wk, R.parieto-occipital subdural, multiple R. frontal contusions. Acute admission 1 week, rehab admission 6 weeks. Independent with self-care and mobility. Performed reasonably well on neuropsych

Case- referral

- Recovery from TBIs complicated by comorbid psychiatric issues, which culminated in a major depressive episode and prolonged admission to the mental health inpatient unit last year after presenting with suicidal intent and ?psychotic symptoms. Antidepressant, changed antiepileptic and antipsychotic medications
- Community MH case management, referred to BI Psychiatry to identify strategies that could further assist in his rehabilitation given lack of motivation to attend to his hygiene, morbidity associated with his malnutrition, low mood and focus on losing weight to the exclusion of other goals
- Main issues client identified- inability to engage in usual activities: riding a push bike, driving, going to shooting classes, walking his dogs and difficulty reaching a previously attained specific weight
- Anticipated accommodation issue d/t mother's ill health

Case- symptoms

- Low energy and motivation levels- unless there is something with a strict deadline, does not act on it
- Does go for walks most days but disappointed only 1 hr, prior to accident walking 5hrs/day and riding 80km to maintain weight. 200kg prior to being made redundant, down to 75kg, gained 30kg during MH admission
- Reasonable mood- sad about 2 dogs
- Listed things he would like to do- looking for a job at a help desk, obtaining amateur radio licence and attending the dentist, but finds it very difficult to follow through with these plans v before TBIs
- Early morning wakening, back to sleep- until 3pm on weekend when no carers
- Denied suicidal thoughts- mother and sister protective

Case- developmental and family

- Quiet, reserved child overweight through schooling years, never any intimate relationships. Felt like he always tried to make his father proud but no matter what, ended up being a disappointment
- Worked excessively- would access system at home, prided himself on being able to solve difficult technical dilemmas
- Father had gambling problems, passed away with renal cancer. Client thought father might have taken excess sleeping tablets to trigger his death in the palliative stages

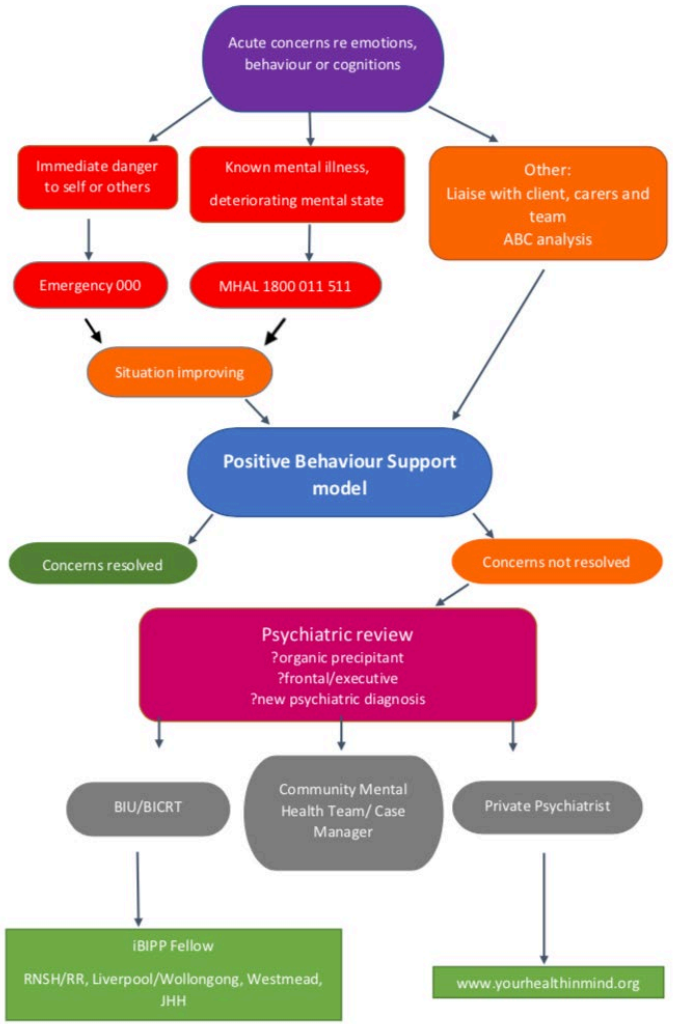
Case- mental state examination

- Caucasian male looking older than stated age. Woken when we attended his house at 10 am and took about 10 minutes to emerge. Casually dressed, smelt of aftershave and his hair was curly and fashioned away from his face. He had long nails which had not been tended to and mild central obesity. He did not make any eye contact and was observed to clench his jaw throughout.
- Walked slowly with a kyphosis and did not seem to pay close attention to cars as we were crossing the road. There was a reasonable rapport formed. There was a latency in his speech which had a slow rate and was relatively monotonous. His mood was described as occasionally dysphoric and his affect was congruent and restricted. There was no formal thought disorder.
- Preoccupation with losing weight. There seemed to be some magical, childlike thinking around connections between his behaviours and weight loss, but they did not appear to be fixed false beliefs that were delusional in nature. Experienced auditory hallucinations on two occasions which were in the form of a public service announcement, prior to his seizures. He reported that throughout his life he has had infrequent episodes of increased sensitivity to smell. These are short lived, self resolve and do not impair his functioning. He has not experienced other perceptual abnormalities. Occ suicidal thoughts but denied current intent.
- Partial insight into his situation with an understanding of the need to see psychiatrists and take psychotropic medication, however found it difficult to make links between his behaviours and his physical issues as well as practical ways to address his amotivation.

Workshop

1. What are your primary concerns about the case?
    1a. What are the priorities?
2. How would you proceed?
    2a. Who do you have at your disposal currently to refer to?
    2b. Ideally what support would you like?
    2c. How does this fit in with the referral pathway proposed?

Draft Proposed Referral Pathway for Brain Injury Clients to access psychiatry



MHAL= Mental Health Access Line, ABC= Antecedent, behaviour, consequence, BIU= Brain Injury Unit, BICRT= Brain Injury Community Rehabilitation Team. Positive Behaviour Support model reference: https://www.icare.nsw.gov.au/-/media/icare/unique-media/treatment-and-care/forms-and-resources/positive-behaviour-support.pdf/?la=en&hash=80E9CC0C543C077BEBC344BDA8EECB74CE4C1E53

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Case- impression

- Significant avolition and amotivation, alongside intermittent low mood and suicidal ideation. Likely related to ongoing depressive episode with perpetuating factors including reminders of his two dogs and difficulty attaining his ideal weight. Background of extensive organic pathology with a stroke and two brain injuries affecting attention and aspects of executive functioning
- Also presenting with a restrictive food intake disorder, present premorbidly following childhood obesity. Long term personality seems to be one of rigidity and obsessionality with a tendency to perform activities to excess.
- Biological vulnerability with gambling issues in his father. Additionally relationship with his father was significant for it's perceived inability to guide and nurture and many of his focuses in life have been to achieve goals which he believed would make his father proud, to no avail. Whilst he desired a partner, he has never been in a relationship and has an underlying low self-esteem exacerbated by his continued need to live with his mother. Long-standing suicidal thoughts with no current intent.
- Ongoing adverse medication effects including bruxism and tremor. May be related to venlafaxine but a trial of reduction in the past was unsuccessful in reducing the symptoms and resulted in a deterioration in mood.

Case- plan

- Cross titration from sodium valproate 500 mg bd to lamotrigine by reducing sodium valproate by 100 mg bd every two weeks and increasing lamotrigine by 12.5 mg every 2 weeks. Nb possibility of Stevens-Johnson syndrome and need to be alert for the development of a rash. CM will monitor for any development of a rash and if this occurs lamotrigine needs to be ceased immediately.
- Consider psychostimulant. Armodafinil 50 mg daily is a reasonable first choice, with the possibility of methylphenidate if this is not effective. Cardiovascular monitoring, contraindicated in the presence of ischaemic heart disease or arrhythmia.
- Liaised with his community psychiatrist
- Psychosocial- meeting with his mother to look at obtaining a dog, liaison with his shooting centre to consider volunteering without accessing a gun and assistance in looking for work. Continue weekly clinical psychology sessions

Panel

Bianca Middleton- Case Manager

Suzanne Stacey- Recreational Therapist

Amy Robinson- Physiotherapist

Joe Hanna- Senior Neuropsychologist

Janine Mullay- Speech Pathologist

Case- update

- Mother passed away, grief with increasing suicidal thoughts
- MH inpatient admission, transferred to older adult ward
- Course of ECT with improvement in mood but cognitive adverse effect
- Accommodation options being explored