THE SOLITAIRE ANARCHIST

Or, The Patient Activist.

by Mark H

Those of the rabble-rousing persuasion, particularly those who lean left and/or who are antistatist, antifa, woke, etc. often find themselves stuck in the clink. In many cases, Uncle Sam / the Guv / Boss Hogg have provided things to pass the time, but not always, and in particular solitary confinement can be a strain on the brain. So this zine collects a variety of card games for one (Patiences, of which Solitaires are considered to be a type, though others would classify the other way around; superfluous semantics, really). The author has played & won (eventually) them all and found these to be a cut (ahem) above the rest (cognoscenti claim some 150 exist). Following these are a few 2 player games for those who find themselves blessed with a simpatico cellmate. If the market doesn't make the cards forthcoming, various DIY's are often available - beta testing for this zine was milk carton-sourced, actually.

Portraits of anarchists and activists have been interspersed throughout. My intention is not at all to trivialize the contribution to the struggle for empowerment & against oppression made by these individuals (plus a collective at the end). Rather I wish to remind the reader that many of them did their own stints behind bars. Don't know if they were Solitaire / Patience players but do know that not on the agenda was the stultification program of their captors. Instead, a mind kept spry meant a soul rededicated to their cause. So enjoy and remember even when you are



HENRY DAVID THOREAU

THE IDLE YEAR

Turn top card from stock. Then the next card. If it matches the first played in rank or suit then it goes on top of it. If not, it's laid down next to first. Keep going in a line. Besides most recent going on neighbor card, two cards away may do same if suit or rank matches but priority goes to neighbor. So here moving 2 over 1 takes priority over moving 4 onto 1. You win if you get down to one stack. It took me a long time to do so though not the year of the title. But my cellie beat it on the second try!



KATE SHARPLEY

THE CLOCK/HIDDEN CARDS/4 of A KIND/SUNDIAL/ALL 4's/TRAVELLERS/THE HUNT

is fairly well known. 13 FD piles will be dealt of 4 each. These 12 around a circle-corresponds to the hours of an analog clock (remember those?) while center is the cuckoo. Turn over top card there. Say it's a 2. Put it on top of cards in 2 o'clock position, FU. Then draw an undercard at that position, move it to its proper hour. A Q goes at noon, J at 11 and K goes in middle. If you turn up all around around the clock before turning up the 4th K in the middle, you win. If not, the cuckoo's caca is on you-you!

ACES UP/IDIOT'S DELIGHT

Deal 4 cards FU. If 2 or more cards in the same suit, remove the lowest card or cards (only the highest stays). Deal 4 more over what remains and remove lows again. If a space is created, a contiguous card may be moved to it. You win if you get down to only the 4 Aces. Difficult to beat but it does delight me. So that makes me....?



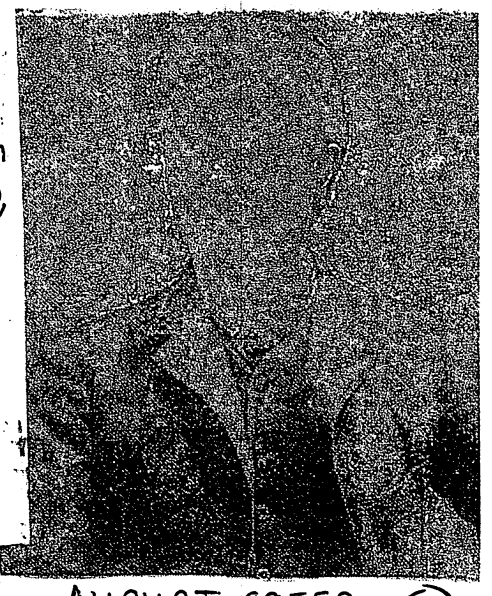
ETER
ROPOTKIN

QUEEN'S/KING'S AUDIENCE

4 FU dealt to each side of a box. From these walls or from stock Aces go inside as turned up but must go w/ J of same suit, just as Ks go inside but only w/ Q of same suit. J serves as Foundation of that suit down to deuce. If can't go inside right then put on waste pile, fill wall gaps with waste pile cards. No redeals but you should not need 'em - it's easy, to beat the Tsar. Eh, Peter?

THE SIEGE FAMILY

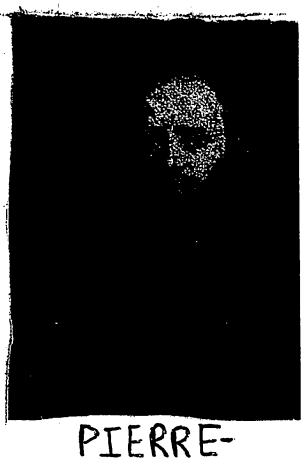
I am not particularly "gung-ho" about any of these, and not just because of their association with the military(-industrial complex), they are just often hard to get going and I like to at least make a little progress before I have to abandon ship once I've dealt. But maybe you'll have better results. In BELEAGUERED CASTLE deal 4 stacked rows of 6 each, most recent on right, all FU. Remove any A's as dealt.



AUGUST SPIES (3)

Do this again with another set of of 4 rows, leaving a column between. There A's will go as Foundations. Found up to K in suit. In the rows one card may be moved at a time to descend, disregarding suit. An empty space may be filled by any card. I think this would be more likely to succeed if entire suit sequences could be moved but it didn't say that in my rules. SELECTIVE CASTLE is imy opinion a little better because after dealing you choose your Foundations' rank (after dealing the 48 and replacing the removed Founders with the left-over 4). FORTRESS is similar to B.C. but it's 5X5 with a column in between, put one of the extras at end of each 1st row. A's are foundations, built up to K in suits. Unlike B.C. end rows may be descended or ascended but one direction per row.

CHESSBOARD/FIVES is F. but Fdtns. need not be A's. Before I sign an armistice on the war theme, one more: NAPOLEAN AT ST. HELENA/FORTY THIEVES This is a 2 decker. Deal 10 stacked rows of 4 each FU. Remaining form stock. Turn up stock one at a time to go to rows or Fdtns. which are the 8 A's placed above as available at row end. Only the end of row card can move and row placement ascends and follows suit. A stock that can't be rowed or founded goes FU onto waste pile. Top of waste always available. K may not descend upon A because A immediately founded. Clear row, any available card goes there.



PIERRE-JOSEPH PROUDHON

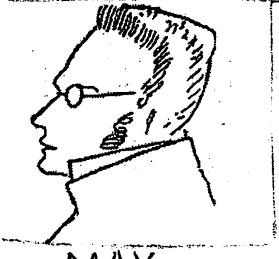


HAKIM BEY

THE SPIDER FAMILY

is led by the popular SPIDER which is very difficult unless you play suit doesn't matter. It's a 2 decker. Deal 10 columns of 5, most recent on top First 4 cards in each FD, last FU. Goal is to assemble 8 columns, A as base ascending to K by suit if you are playing that way or not if you're not. You may move one card to ascend onto another at row bottom, but you may also move a correct ascending sequence but not if it has any out-of-seauences on top of the in-sequence ones. A K may only be moved onto a columnar gap which may however ... be filled by a unit of movables (I take this to mean no non-K singletons). When moves at a standstill deal 10 more FU at column bottom. Gaps must be filled before these deals happen, unless you are playing "relaxed spider." In WILLO' THE WISP you use 1 deck with 7 columns of 3 each. First 2 are FD last is FU. SPIDERETTE also uses 1 deck but its layout is that of Klondike, which it is tougher than even if you're disregarding suit (generally because the new deals stop movement unlike Yukon or Russian Sol). SCORPION has 7 columns like WotW but 7 in each, 4 FD then 3 FU. 52-49=3 which become reserve, they get dealt after initial moves made. Unlike Spider, any FU singleton or group in or out of sequence can move. Turn FD FU if they become available. As with Spider, empty columnar gaps filled with K or any group. Win is 4 in-suit seauences K down to A so this is as difficult as Spider due to suit mattering but at least out-of-sequencers don't block moves.

LITTLE SPIDER is in the family in name only. A's low. 4 cards FU in a row then 4 more below also FU, but leave space between for Founders. These a red K & black A. Then the other red A & the other black K. The A's will found up to K, K's down to A (in suit of course). Any card may drop from top row to found. But bottom row card may only rise to the Fdtn directly above it. However, when deck exhausted, bottomer may rise to any. Also top cards from layout may stack up or down upon each other, suit not mattering (and for this A may be followed or preceded by K). Keep covering rows with new deal. You cannot fill spaces so refrain from making any. I've run out of space for MISS MILLIGAN (not Muffet) but it's a 2 deck Spidery Klondike with "weaving." Sorry!



MAX STIRNER

What the non-cognoscenti know as Solitaire is generally KLONDIKE. It's 7 FD across then 6,5, etc leaving 24 in a draw stack. Now if you take those 24 and play them FU, evenly distributed on the columns except for the singleton on the left, you are now in the (Arctic) territory of two other (IMHO better) games: You can now move any of those columns to other columns to open up cards to go up to Foundations (Aces like Klondike). If you're just descending rank in alternating colors, this is YUKON. If you're descending in rank & suit it's RUSSIAN SOLITAIRE which is my favorite of the 3 though it's very difficult to beat.



NOAM CHOMSKY

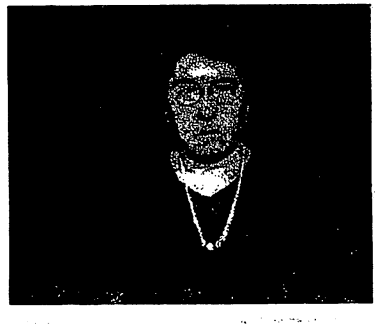
THREE BLIND MICE (5) is in the same family as Russian & Yukon. Deal 7 FU, in that same row deal 3 FD. Do this 2 more times below 1st row. Then deal 2 more rows of 10, all FU. Align in columns as in R.S./Yukon. The remaining 2 cards are dealt separately FU as a reserve. Move columns as in R.S. (descend in suit & Ks go up to fill empty columns). Reserves may be played appropriately but reserve space is not refilled. A win is four sequences down in suit from K to A, so this is easier than R.S., but fun!

LA BELLE LUCIE/MIDNIGHT OIL

I'm not a big fan of this but it was very popular back in the day and it does play fast. Deal 17 FU fans, most recent on top, on right of each fan. Found A's as they become available. Top right of each fan is available to found on A to K in suit or move to a top right to descend in suit. You get 2 redeals in which a reshuffle (a rarity in sol) is allowed in each. Apparently only singletons are allowed to move to another fan (including the singleton left by dealing 17x3=51). A win could be facilitated by allowing also in-suit sequences to be moved? (THREE SHUFFLES & A DRAW allows one buried card to be "rescued" to the top of its fan on the last deal. BTW)

TAM O'SHANTER

Four FU in a row. As A's come up, play above as Foundations. Suits disregarded to Found. Keep dealing series of 4 FU. If you pull the A's as Fdns. before you deal, then it's AULD LANG SYNE. Cheers!



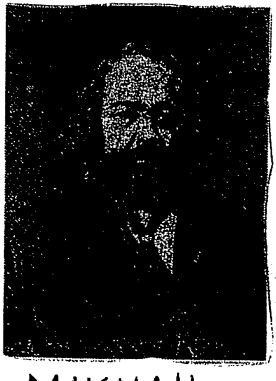
EMMA GOLDMAN



MARSHA P. JOHNSON

PYRAMID

Pretty well known but I will present it anyway. A single-tan FU way up top. Then 2 below then 3 etc ending w/ 7 at base. K's=13 and are removed from base. Also removed are any pairs that sum to 13; Q's=12, J's=11, and A's are low. Put removed into FU pile top of which also available to pair up again. Rest of stock dealt to pair w/ lowest row of pyramid. If can't, go to waste (not removed); 2 redeals of waste pile allowed. Various scoring schemes, but you win when no pyramid, essentially.



MIKHAIL BAKUNIN

ROYAL PARADE / FINANCIER / HUSSARS / THREE UP

(Yes, more elitist titles but we will just consider the first in honor of Ms. Johnson, okay?) This is a 2 decker. Deal 7 FU cards in 3 rows. As a 2, 3 & 4 come up, place them respectively next to the 1st, 2nd, & 3rd rows. Immediately fill their spaces in rows. If you haven't dealt out the 2 3 or 4 by the end of making rows go and pull them out now. On the "2" row will also be located 5's, 8's, & J's. On the "3" row will also be located 6s, 9s, & Qs. On the "4" row will also be located 7s, 10s, & Ks. Go through and first turn sideways any cards which don't belong in their rows. These can then be traded for the right cards in other rows. Aces are immediately discarded. Below these is a row of 8 FU. Whenever play is stuck deal 8 more ontop of these. Your goal is to build up foundations by suit, up in rank but a card has to be in its home row before it can be stacked. (So on 2s row for instance 2♡ on base, 5 over it, then 8, then J♡, on top. It would be imprudent to build on any but the 2, 3, or 4 unless you are playing that "unstacking" can occur.) Keep moving up bottom row cards. I sideways the 2nd & 4th in each stack as a visual cue. It is also advantageous to vary the suits you move up to the Foundation rows. It is also better to wait and fill a gap with a lower card especially if you aren't allowing unstacking. I turn each completed stack over. You win if you have 3 rows of 8 stacks each. Chances of winning are @ 1/8, slightly better w/ unstacking, but it can put you in a bind too!

(VIRGINIA REEL is supposedly easier than RP though my description of RP removes some of RP's kinks, so I don't feel the need to go into V.R. here.)

KNOWLEDGE

by Martha Nov. 2019

What do you do when you feel your brain is not functioning lik it normally does, causing you to question if the symptoms tha you perceive as real are not just "in your head" but also impacting the nervous and other body systems? This occurred me not too long ago and I can't say whether or not I'm over it, with the stressors that may or may not have caused what v possibly a "nervous breakdown" still being present. Or mayb It was virally-initiated; I may never know. But what's mos important about it is that it caused me to re-evaluate what I thought I knew as a layperson about the nervous syster and its interaction with other body tissue and fuel. I was reminded that it is fundamentally unhelpful (and maybe unhealth too) to think of the mind as separate from the body. So trying to frame this (continuing?) crisis as a gift to spur action rather than a hinderance, I committed myself to doing the research I needed to do to get a basic grasping of psycho-neural anatomy and function and the neurotransmitters that make the system dy amic. As an inmate, my ability to do so is limited but Lexis-Nexis and the book The Traumatized Brain got me started. The rest of the task was realized by significant Wikipedia printing by my mother, for which I am grateful beyond words. Inmates like myself know how frustrating it is to r ceive what is often subpar medical & psychological care ar disturbingly frequently, deliberate misinformation. So this zinelet is intended mainly for an audience of the detained wi access to the Web. It is by no means a substitute for the advic of a medical professional, but I hope it'll be helpful to those who are suffering or maybe just curious as to how this stuff works, a least what will fit onto 24 pages. Inmates like myself who attempt to retain some sense of autonomy must bear the scorn often of b the administration and [illegible]

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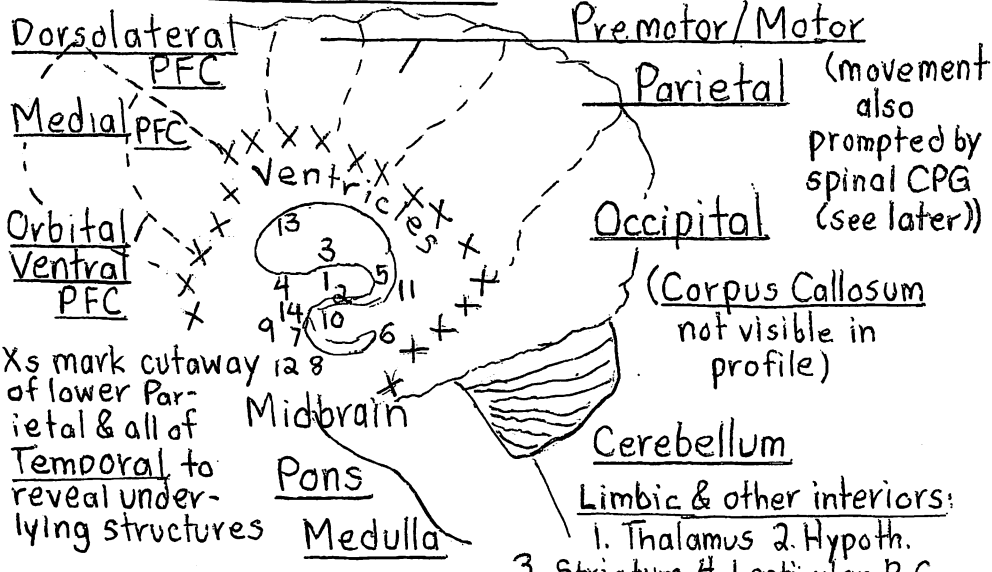
But I remain antifa, antiracism, and queer and will keep learning as long as I can because a mind is too much of an amazing thing to be wasted! This what I've recently gathered about it:

ANATOMY/FUNCTION



ORBITOVENTRAL PREFRONTAL CORTEX "The Ego Area." Hyperfunction here can = self-centering, repetitive thoughts. Seizures here can = "forced thoughts." Damage here can = aggression. Broca's in lower region is a speech center (see APHASIA in CHALLENGES).

MEDIAL PFC contains the ANTERIOR SINGULATE Bridge between the "feeling/reacting" areas of the limbus and the centers of "higher" thought. Fosters selective attention & choice. Detects errors and resolves conflicts. In DEPRESSION its subgenus is believed to be hyper- allowing too much input from emotion centers. One study found that a larger A.S. correlated with greater political liberalism. In the limbic circuit the A.S. receives signals from the thalamus and sends to the hippoc.

DORSOLATERAL PFC "Just done... doing... will do." Center of abstraction, inhibition, & task-switching. Working (prior to short-term) memory here. Hypo- in DEPRESSION, probably a cause and effect.

CORPUS CALLOSUM connects the hemispheres generally from OVPFC (especially Broca's) in front of the

limbus and all the lobes aside & behind it. Larger in females and the ambidextrous/lefties. Under-developed often in dyslexia and autis Musical training increases its plasticity.

INTERIOR STRUCTURES of the LIMBUS

RED NUCLEUS is actually pink, due to hemoglobin. In babies controls crawling. In walkers, arm swinging. Some hand control als

HYPOTHALAMUS - Controls thermoregulation via shivering a fever. The hippoc. and amygdala project to it, driving its control of th autonomic nervous system, via the medulla. The hypoth. projects t the cortex via the thalamus and to the pineal via neck sympatheti nerves. It also projects to the VTA (see below) to excite it. In response to nipple-sucking promotes pituitary PROLACTIN (see CHEMISTRY). Prompts pituitary FSH & LH (sex hormones) by secreting GnRH. Produces OREXIN as part of its control of hung & feeding. Controls thyroid via feedback. It fosters NREM slee while its suprachiasmic nucleus is body's "bioclock" by VIP(se GABA & OREXIN et al. A rage center. Unlike most other brain structures, partially unshielded by BBB (see below).

AMYGDALA - is bicameral, like mamm. bodies, thalamus. Left produces happiness as well as fear & anxiety, and sadness, while right is solely negative, thus prompting aversion. However, fear may still be processed w/o a functioning amygdala. Right is also linked to declar tive (consciously recalled) memory, as well as episodic (emotional/ sensory experience not requiring conscious recall). Rich in androge receptors, biological males' are generally larger than females' afte puberty. Gay men demonstrate more female-appearing amyg. tha heterosexual men while lesbians' are often more male-appearir Young children's amygdalae react differently to opposite gender th the same. This declines until puberty when it increases dramatical again. It projects to the hypoth, thalamus, trigeminal & facial ner es, the VTA, and the basal ganglia nucleus accumbens. It receive sensory input from various senses, especially the olfactory bulb. Al an appetite center like the hypoth. Prompts threat responses such defending or escaping, ↑bp, and heart rate, and stress hormone rele ase. It also responds to pheromones. Its connection to the nu leus accumbens involved in motivation/reward. Buddhist monks prac icing metta (compassion) meditation showed ↑ activity here. Lesions here may produce loss of fear and familiarity. Social phobia patients sho ↑ed response here. Amygdala volume positively correlates with size of one's social network. One with a damaged amyg. often lacks response invasion of personal space. Stimulating the amyg. ↑s both sexual an aggressive behavior. Enzyme PKC-ε here regulates behavioral response to ethanol & morphine. One study found that increased vol in its right hemisphere correlated with political conservatism.

INSULA Expert meditators showed more activity here than novice: But is also a craving center - Smokers with damage here lost their urge to smoke. Maybe the meditators are "addicted" to serenity?

MAMMILLARY BODIES - Relay impulses from amygdala and hippoc. to thalamus. Important for recollective memory and spatial recall. Lesions here may produce amnesia. Poorly understood but implicated in many abnormal conditions (see CHALLENGES).

ENTORHINAL CORTEX is the gateway to the HIPPOCAMPUS which connects with the hypoth. via the FORNIX. The hippoc. compares stimulus to memory. The limbic circuit can be considered to begin here, a memory impulse then goes to fornix, mammillary bodies, thalamus, PFC cingulate, entorhinal, and back to hippoc. Stress hormones like corticosterone shrink it thus the memory issues in PTSD. et al. One of the first areas affected by altered blood flow. Damaged of course in Alzheimers. Smaller than normal in depression and SCHIZOPHRENIA (see CHALLENGES).

BASAL GANGLIA contains many distinct substructures including the putamen, caudate, globus pallidus, STRIATUM, and nucleus accumbens. Sends motor info through "attention circuit." Receives amygdala impulses which are glutamine-driven and depend on dopamine D1 receptors to drive "reward." In its nucleus accumbens, dopamine response is sensitized by stimulants and sexual intercourse but not by opiates or high fat foods/sugar. Stimulants and sex also increase nerve quantity here while opiates ↓ it. Striatum opioid receptors are generally increased by all the above (see ENDOGENOUS OPIOIDS in CHEMISTRY).

THALAMUS receives input from hypoth. via mammillary bodies and outputs to cortex via the singulate. Also a bridge between the skin and the parietal lobe, integrating its tactile info w/visual and auditory. Thus it is a spatial attention center. It is bicameral: left hyperfunction = anger, right associated with mania, confusion.

VENTRAL TEGMENTAL AREA/SUBSTANTIA NIGRA are distinct areas with similar function. The S.N. projects higher into the B.G. than does the VTA, both being dopaminergic centers. VTA receives impulses from PFC, hypoth, basal ganglia, and the HABENULA at its rear. The first 3 are largely glutaminergic "Go pedals" while the habenula is the GABAergic "brake." VTA outputs to those first 3 also, as well as to the amygdala, hippoc, & olfactory bulb. VTA is implicated in driving both obsession and "love," as in animals it regulates pair-bonding. Besides reward, the VTA connections to the striatum and olfactory bulb drive aversion. Unsurprisingly, along with the B.G, the VTA drives drug addiction and, interestingly, foreplay.

The following 2 glands are cerebral and thus discussed here. (See CHEMISTRY for the THYROID & PARATHYROID glands.)

PITUITARY GLAND - aka Hypophysis. Produces PROLACTIN (see CHEMISTRY) mediated by the hypoth. Prolactin feedback regulates pituitary production of the sex hormones LH (for sperm production) & FSH (for ovulation). See PACAP in CHEMISTRY.

PINEAL GLAND-Located between the halves of the thalamus behind t habenula. Size @ grain of rice but second only to kidney in blood flow, which is monitored by phagocytes. Unlike most brain structures, it is not shielded by the BBB. Connected to both the symp. & parasymp. N.S. as well as the trigeminal w/ neurons containing PACAP. Its metabolism led by Endothelin-1 (see CHEM.) May influence actions c rec. drugs like cocaine and SSRIs. Produces melatonin and possibly pinoline. Paradoxically, children produce more mel. than adults, who tho of course have larger pineals. Receives light info from hypath. - some reptiles & amphibians still get this from their "pineal (third) eye." Also infl uences pituitary secretion. Frequently calcifies and fluoridifies with age. Aka epiphysis. (see BRAIN SAND)

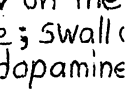
FLANKING/REAR/CAVITIES/OTHER

TEMPORAL LOBE- Visual & auditory processing, also declarative m ory. Anterior damage here can = loss of fear & regret, ↑aggression, hypersexuality, hyperorality (inappropriate objects in mouth). Sei- zures here = deja vu, hallucination, "rollercoaster" rising from abdo- men. Wernicke's is in its upper region, a speech center (see APHASIA)

PARIETAL- Integrates spatial sense and navigation. Haptic (skin) touch, temp., and pain impulses travel here through thalamu Also a math center. Whereas temporal carries "what" of vision from occipital, parietal is responsible for "where/how." Sends t motor area arm, hand, eye commands. Also receives vestibular input to control balance. Damage here in the right hemisph. = loss of imagery, spatial visualization, neglect of left of body. Left hemisph. dam age = math, writing and symbol recognition↓. General damage may = sensory seizures, dyslexia and anosognosia (a physically challenged individual seems unaware of it). Left damage may also prompt agnosia (full body los of perception). May play role in predisposition toward spirituality.

OCCIPITAL- Eyes to thalamus, via optic nerve, hypath. Thalamus co verts l-r/r-l and sends here which sends to temp./pariet. Seizures h = visual hallucinations.

CEREBELLUM- Animal studies suggest its involvement in arousal, feeding and predation. In humans clearly involved in fluidity of thought/speech, balance/coordination and movement.



PONS- Activates REM sleep, paralyzes motor during sleep.

MEDULLA- Autonomic functions (breath, heartbeat) & CPG (see belov

VENTRICLES- 4 areas of fluid. With age they increase, brain shrink

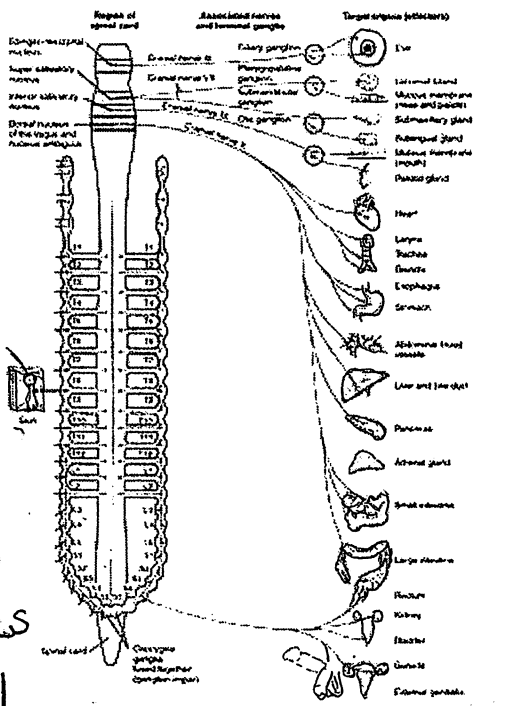
CENTRAL PATTERN GENERATORS- In theory a human can run ar- ound like a headless chicken. Activity here doesn't rely on the cerebral cortex. Locomotor ones are in base of spine; swall ing, chewing and respiration in medulla. Mediated by dopamine serotonin, and tachykinin (see NEUROKININ) et al.

AREA POSTREMA/CHEMOCEPTOR TRIGGER ZONE- Just outside BB "Need to vomit" signal from blood (or from stomach via VAGUS) travel

ack down vagus to cause contraction to vomit. The CTZ is very near he SOLITARY TRACT NUCLEUS which is the portion of the medulla where LL visceral information is integrated. Conditional taste aversion (the nemory that ensuresan animal will not resample a toxin) also arises here. BBB - the Blood Brain Barrier which protects it from toxins and other blood-borne langers; however for a medication to have any cerebral effect it must be able o pass through the BBB as well (noted earlier are the excepted Pineal & Hypoth.)

AUTONOMIC NERVOUS SYSTEM

consists of the sympathetic and parasympathetic systems of visceral nerve connections. It s regulated by the hypoth. acting n response to limbic and visceral sensory info. There are 3 receptors, for NOREPINEPHRINE, ACETYLCHOLINE, and NITRIC OXIDE (see CHEM.) Nerves going TO the organs are Efferent, ones coming BACK to the spine are Afferent. Sometimes one nerve bundle may serve both functions.



SYMP. nerves emerge from the horacic and lumbar spinal areas. Between the spine and organ is generally a pair of nerves, the 2nd of which is "splanchic." The exception to this are the ADRENALS which are directly innervated (see following).

The symp. vasoconstricts to draw blood from the gut and skin to the lungs and muscles. The lungs dilate due to EPINEPHRINE circulating in blood (why it's administered in anaphylactic shock). Pupils dilate, far vision is enhanced. Intestinal sphincters constrict, peristalsis inhibited. Urinary bladder relaxes. Heart rate increases. All the above = "fight or flight." Symp. also stimulates orgasm.

PARASYMPATHETIC SYS.

PARASYMP. nerves emerge from the cranium and the sacrum as visualized above. Cranial nerve III is ocular. CN VII is submandibular (under jaw) and pterygopalatine (tears & mucosa). CN IX is otic (but see below). CN X is the VAGUS, a major afferent & efferent pathway. Parasymp. increases blood flow to the gut, contracts lungs, constricts pupils and enhances close vision. It stimulates saliva as well as the pancreas and gallbladder. Gut peristalsis is activated. It contracts the bladder and prompts erection and other sexual arousal signs. All the above may be considered "feed and breed" or "rest and digest."
"Vasovagal maneuvers" like coughing, holding breath for a few seconds,

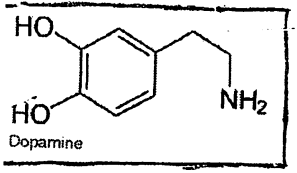
and pressing as if to defecate can stimulate the parasymp. (see also DYSAUTO NOMIA in CHALLENGES). E

CAROTID BODY This small but significant region of the carotid artery is innervated by the CN IX. Blood levels of oxygen and CARBON DIOXIDE are monitored here.

ADRENAL GLANDS (above Kidneys) - Their cortices' connections through feedback to the hypoth. via the pituitary are the most relevant of their many functions to this zine. Besides activating the symp. just discussed, stress/fear prompts the hypoth. to release VASOPRESSIN and CRH. These in turn prompt pituitary ACTH secretion. ACTH acts upon the adrenals. The adrenal cortex then produces the stress hormone CORTISOL (among others) while the adrenal medulla produces EPI/NOREPI. Vasopressin (which is a potent vasoconstrictor) may be also ↑ed by dehydration as it's the "water-conserving hormone." Also produced along with ACTH are Beta-endorphins. In the healthy cortisol spikes after morning rising, then it falls only to spike again at supper time, then falling until midnight (an evolutionary hunt-gather/rest cycle). This cycle disrupted in insomnia as well as chronic fatigue. Cortisol also regulates the immune system, thus corticosteroids are given in allergic & inflammatory processes. But chronic stress has been found to both hyperactivate and suppress immune response and thus more study needed there. OXYTOCIN (see CHEM.↓) suppresses cortisol and thus counteracts stress and promotes wound healing. Cortisol shrinks the hippoc. and this effect on PTSD has already been noted but paradoxically PTSD patients show blunted cortisol release. Prenatal stress resulting in ↑ cortisol has been implicated in childhood ADD & depression. (see also PHEOCHROMOCYTOMA in CHALLENGES, ALPHA/BETA in CHE

CHEMISTRY - Prison medication regimes tend to be the cheapest they can find but free info's a powerful therapy too. Here are the major neurotransmitters as well as some interesting minor ones, as well as major classes of psychoactive medications. Wish I could present them all but an 80 page zine seems a little excessive, right?

The fundamental chemistry of every neuron is: Na (sodium) or K (potassium) enter to negativize it. When negative enough it fires. Such firing is hyper- in epilepsy et al. SODIUM CHANNEL BLOCKERS this Na intake. Also pervasive is ionic calcium CA^2. There's usually 10000X more outside a cell than in. Calcium channels open to decrease this ratio. There are four different types of these; CALCIUM CHANNEL BLOCKERS generally prefer the L-type which is that found on the adrenals just discussed. There Ca influx produces ALDOSTERONE, thus ↑s. Thus CaC blockers like Amlodopine/Norvasc ↓ bp. They also are vasodilators slowing down heart rate & force. N-type channels are those of the nerves so their blockers find use as antiepileptics and antineuralgics. They include Nimopodine/Nimotop, Lamictal, Lamotrigine and Ziconotide which is 1000X more potent than m



DOPAMINE - 5 confirmed receptors with 6&7 speculative. D1 and D5 in one family, D2-4 in the other. There are many more D1 & D2 receptors in the brain than the others. D receptors aren't just in the brain, other locations noted if significant. In general D is a vasodilator. It is involved in vomiting, thus its blocker Metoclopramide/Reglan is antiemetic. Involved in schizophrenia (see CHALLENGES) but exactly how is still debated. Implicated in Tourette's and most certainly involved in Parkinson's (which see). "Typical" antipsychotics are solely dopamine antagonists while atypicals block serotonin and other neurotrans, also. Abrupt cessation of D ↑ers has been implicated in neuroleptic malignant syn. (which see) along with use of D receptor blockers. Blockers like: Risperidal, Sulpiride, and the atypical antipsychotic Iloperidone/Fanapt and many others, can produce muscle tremor and slowing (tardive dyskinesia) because D is key to Basal Ganglia's motor function control in addition to its reward-stimulus. D↑ers which are used to treat Parkinson's and ADHD and other challenges include Amantadine/Memantine/Namenda, Bromocriptine, Modanifil, Cabergoline Selagiline/Eldepryl, Carbi/Levadopa/Sinemet, Wellbutrin, Metadate, Rasagiline, Ritalin etc. Another blocker is Lacosamide/Vimpanil.

D1 - Activates MTOR. Also in heart, kidney. Addictive drugs like cocaine & meth inhibit the D transporter, making it more available and thus ↑ing # of D1 receptors, driving B.G./VTA reward system. D1 most implicated in D-induced ↑ bp. D1 stimulants do initially ↑ cognition but habitual high dosing shown to promote cogn. deficits. High fat/sugar foods ↑ D1 signalling in the B.G. striatum. Blocked by Seroquel/Quetiapine, et al.

D2 - Also blocked by Seroquel et al. Also found in heart, kidney. While D1 ↑ in addiction-reward, D2 is ↓. Defective version assoc. w/ compulsive gambling. Cocaine, meth, heroin, alcohol all ↓ it but aerobic exercise and environmental enrichment ↑ it. Its blockade seems to produce massive glutamate release.

D3 - Mostly in brain. In B.G. ↑ed by cocaine, opiates and fat/sugar.

D4 - One of its varieties most strongly implicated with ADHD. Blocked by Clozapine/Clozaril et al.

D5 - See entry on BDNF.

D1&2 also like Adenosine Recep.
D4 like Alpha 1B & Beta 1 (see later)

mTOR - Studies on this enzyme exploded when it was ↓ed in worms and mice and they lived longer than normal. Accordingly its ↑ing prompts many types of tumor growth. Besides D1 above, it is ↑ed by the ingestion of leucine and other amino acids and therefore is associated with muscle growth. Caloric restriction ↓s it and thus lateexpands human lifespan. It proteocomplexes to form mTOR 1&2, with 2 being shown to promote nerve growth but also "brain

trash" like amyloid plaque and tau protein (see ALZHEIMERS) and huntingtin in Huntington's disease. In the lab, curcumin and caffeine ↓ it but this hasn't been replicated in humans. Berberine was found to do so, though. mTOR may be implicated in brown & white fat development.

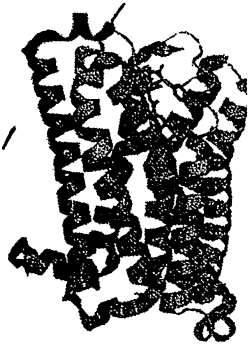
PROLACTIN - Hypoth. secretions prompt dopamine release which act on lactotroph D2 receptors to inhibit prolactin and thus inhibit lactation. Prolactin received in reproductive organs prompts testosterone/estrogen release. It promotes the genesis of cells that mature to coat nerve axons. Interestingly, it promotes nerve growth in both fetus & brain of mother. Its levels peak during REM sleep and in early morning. It inhibits hair growth. It is ↑ed by VIP (which see) and by stress, exercise, high protein meals, MDMA, anesthesia, H2 antihistamines, antipsychotics, opiates (but also opiate antagonists), shingles, kidney failure, cirrhosis, polycystic ovarian disease, and hypothyroidism et al. Anything that ↑s dopamine could ↓ it.

SEROTONIN - While dopamine is primarily excitatory (except for D2 reception) 5-HT is both excitatory and inhibitory. Serotonin receptors modulate the release of many compounds listed in this zine including Glutamate, GABA, dopamine, epi/norepi, acetylcholine, substance P and cortisol, among others. 5HT receptors are involved in aggression, appetite, anxiety, cognition, memory, nausea and thermoregulation. In the well-studied worm C. elegans 5-HT receptic has been even found to modulate lifespan (like mTOR).

Of the dozen-plus subtypes listed below 2A, 2C & 3 find the most use in medicine.

1A, 1B, 1D, 1E, 1F are inhibitory. They are antagonized nonselectively by methysergide (which is therefore excitatory). 1A blocked by Seroquel/Quetiapine et al. but ↑ed by Vibryl/Vilazodone.

2 receptors are excitatory thus their blockers are inhibitory. 2A blockers often find use as atypical antipsychotics and include methysergide and Seroquel as well as Ketanserin, Cyproheptadine (an antihistamine), Clozapine and Nefazodone. Some 2A blockers will also block 2C including Ketanserin, Clozapine, Nefazodone, Risperidone, Trazodone/Desyrel. (2C used to be 1C).



The 5-HT1B receptor as an example of a metabotropic serotonin receptor. Its crystallographic structure in ribbon representation

3-receptors are ion-channels, unlike any of the others. They prompt vomiting thus 3-blockers are antiemetic and include Dola-, Grani-, Ondan-, Palono-, and Tropisetrons and Mirtazapine/Remeron. (see also IRRITABLE BOWEL SYNDROME).

Nonselective 5-HT receptor blockers include Chlorpromazine, Meteroline, Mianserin, Oxeterone, Spiperone, Ritanserin and Propanolol

Receptors 4-7 do not find use in medicine. BUSPAR activates 5HT1A.
TRIPTANS are 5-HT activators which are used to stop in-progress migraines. They include Suma-/Imitrex and Ele-/Zomig/Relepax.
SSRIs make 5-HT more available by inhibiting its reuptake, usually by blocking the SEROTONIN TRANSPORTER. People who produce its long form have more success with SSRIs, which include Fluoxetine/Prozac, Sertaline/Zoloft, Paxil/Pexeva, Citalopram/Celexa, Escitalopram/Lexapro. Sexual dysfunction is a common side effect of SSRIs. To avoid SEROTONIN SYNDROME (see CHALLENGES) they should not be taken with MAOIs (see Monoamine Oxidase). Add norepi to the reuptake inhibition and you get SNRIs which include Luvox/Fluvoxamine, Venlafaxine/Effexor, Desvenlafaxine/Pristiq, Duloxetine/Cymbalta, Milnacipram/Savella, and Levomilacipram/Fetzima. MARIs include the other catecholamines and the appetite suppressant Mazindol/Mazanor/Sanorex is one of them. (See also ANTIDEPRESSANT WITHDRAWL SYNDROME in CHALLENGES regarding the sudden stopping of these medications).
TRICYCLIC ANTIDEPRESSANTS may also prompt AWS. They include Imipramine/Tolfranil, Desimipramine/Anafranil, Amitryptiline/Elavil, Amoxapine/Asendin. Due to toxicity (and AWS) not used as frequently as they once were in favor of TETRACYCLIC ANTIDs. They have 4 rings rather than 3 and include Mianserin/Tolvon, Septiptiline/Tecipul, Benzoctamine/Tacitin, Trazodone/Desyril, and Mirtazapine/Remeron. All except Remeron block norepi reuptake making it more available. But Remeron enhances CNS norepi by blocking Alpha2 reception as does Tolvon (see ALPHA/BETA RECEPTORS). All TeCs are strong H1 ANTIHISTAMINES but are less drying because they don't affect ACETYLCHOLINE (see both later).
The amino acids GLUTAMATE and ASPARTATE (most notably in the form NMDA) are the brain's primary excitatory transmitters. Because sodium channels often prompt their release they are suppressed by NaCBs. Ingestion of glutamate has neurological effects including ↑ing BDNF (see later). Glutamate has its own specific receptors but will also bind to NMDA receptors as will the other amino acids d-SERINE, GLYCINE and ARGININE. Arginine also binds to Alpha2. Opposing Glu and NMDA is the major inhibitor GAMMA-AMINOBUTYRIC ACID (GABA). It lowers anxiety & aggression. GABA ↑ers include Neurontin/Gabapentin, Lyrica/Pregabalin (both also CaCBs - GABA is released through this channel also) and Gabatryl. Some GABAergics may prompt seizures, though (while Neurontin is paradoxically an anticonvulsant). Appears to oppose BDNF. GABA has a circadian rhythm, coactive with VIP (see later).
PHENOTHIAZINES are antianxiety/antipsychotics that also quell nausea. They include Promethazine/Phenergan).

Prochlorpromazine/Compazine, Fluphenazine/Prolixin, Stelazine, Thorazine and Mellaril.

BENZODIAZEPINES are addictive sedating mood stabilizers. They include Diazepam/Valium, Alprazolam/Xanax, Alonazepam, Klonopin, Temazepam/Vivactyl/Restoril, Lorazepam/Ativan and Chlordiazepam/Librium.

PACAP (Pituitary Adenylate Cyclase Activating Peptide) is similar to VIP. Stimulates ECL cells (see ANTIHISTAMINES). Also a neurotransmitter. In women, a defective form of it has been implicated in PTSD so it may promote resilience.

SIGMA RECEPTORS are poorly understood. In some they produce euphoria, in others malaise and anxiety. Sigma blockers maybe considered antipsychotic and have also been used to block convulsions from cocaine overdose. Sigma is activated by Berberine, Citalopram/Celexa, Escitalopram/Lexapro, Cocaine, DHEA, Dextromethorpan, DMT, Fluoxetine/Prozac, Ketamine, Meth, Ritalin, PCP, Pregenolone. Sigma is blocked by Haloperidol, Sertraline/Zoloft, and Progesterone.

ENDOGENOUS OPIOIDS produce the well known "natural high" of exercise and adventure. Endorphins bind to Mu receptor, Dynorphin to Kappa & Enkephalins to Delta. (Beta-Endorphin already discussed in ADRENALS)

NEUROPEPTIDE FF interacts with endopioids to modulate pain. Also involved in cardiovascular regulation, body temp & macrophage activation.

CAFFEINE's effects are well known but maybe not fully perceived by its millions of users. Short-term it ↑s bp and increases sympathetic NS effects. Long term (habitual) consumption however appears to stimulate the parasymp. So it is a vasoconstrictor acutely but is a dilator in "caffeiholics." It can be considered in both cases a stimulant due to its ↓ of ADENOSINE (see later). Another xanthine, THEOPHYLLINE has the same effect.

ANTIHISTAMINES either block mast cell release of H1 in allergy (sinus & skin) or H2 from stomach ECL (enterochromaffin-like) cells (see PHEOCHROMOCYTOMA in CHALLENGES). Ranitidine/Zantac is a H2 blocker. H is also involved in vomiting signalling so many antihistamines like Dramamine but also Diphenhydramine/Benadryl are antiemetic. Most are anticholinergic but Remeron isn't. Most that block H1 are sedating. Antihistamines that also have anti serotonin activity: Carbinoxamine, Methdilazine, Pizotifen, Promethazine/Phenergan and Cyproheptadine which is OTC in Latin America as an appetite stimulant for children. Hydroxyzine/Atarax/Vistaril is also antiserotoninergic and OTC it is a weight loss aid (see also ADHD). Other antihistamines include Astemizole & Mizolastine.

ACETAMINOPHEN acts upon hypoth. to reduce fever & quell pain but it has no antinflammatory effect like aspirin & other NSAIDS do nor does it thin the blood.

MELATONIN- Produced from serotonin in the pineal. It facilitates sleep and helps prevents osteoporosis. Stimulated by dark, inhibited by light.

MONOAMINE OXIDASE It breaks down the catecholamines- D, 5-HT, nor/epi, and H. Block it and they are more available. However, ↑ed norepi can lead to aggression and nightmares; ↑ed H can lead to rhinitis, nausea. ↑ed D may raise bp. ↑ed epi can lead to heart palpitations, so MAOIs should be used with caution, especially with SSRIs, because too much 5-HT can lead to serotonin sickness (see CHALLENGES).

DECONGESTANTS are vasoconstrictors that prefer sinus area. Non-Ephedrine are OTC.

ALPHA/BETA ADRENERGIC RECEPTORS/NOREPI/EPI - Numerous entries here discuss epi (adrenalin) and norepi (noradrenalin) so they need not be discussed separately. Norepi and epi are the endogenous ligands of adrenergic receptors but A2 can also receive Arginine. A1s increase heart contractive force, dilate pupils by contraction, erect hair, and contract prostate all by ↑ing cellular calcium release. A2s aggregate platelets, inhibit transmitter release from adrenergic and cholinergic nerve terminals and fat cell lipolysis, all by ↓ing cyclical adenosine monophosphate (cAMP). A1 blockers include Doxazosin/Cardura, Tamulosin/Flomax, Terazosin/Hytrin, Seroquel/Quetiapine, Ketanserin and Prazosin/Minipress, which has been used against norepi induced nightmares in PTSD. A2 blockers include Yohimbine and Remeron, the former ↑ing bp and heart rate and considered a sexual stimulant, the latter an antidepressant. Blockers of both A1 & 2 include Mianserin/Tolvon, Trazodone/Desyril, Phentolamine/Regitine and Phenoxybenzamine/Dibenzyline, which is an irreversible blocker. The A1 receptor can mimic D4 dopamine receptor, as can Beta-1. Some Beta blockers like Labetol/Trandate and Carvedilol/Coreq also affect A1 receptors and find use against congestive heart failure.

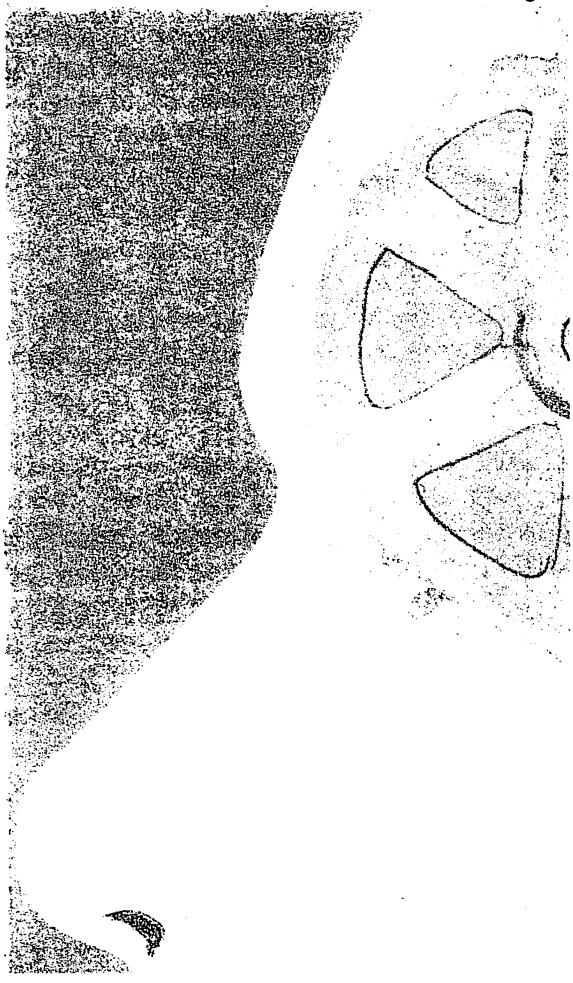
There are 3 types of Beta receptors. B1 are mainly cardiac and renal. -2 are in lungs, GI tract, liver, uterus, blood vessels and skeletal muscle. -3 are in fat cells. B2 agonists like albuterol are respiratory aides. Beta blockers of course competitively antagonize beta receptors. The first to be synthesized - a revolution in cardiology - was Propanolol/Inderal (Black 1964). Its original use was anti-angina and it was followed by a multitude of -lols to treat high BP, though since ACE inhibitors, CaC blockers and diuretics have been found to be more effective in preventing stroke. Propanolol has found subsequent use as a migraine preventative and as "the performer's friend" to reduce the tremors of stage fright (by stimulating the parasymp. n.s.)

ADENOSINE is generally inhibitory. Ad1 and 2A slow the heart and and also modulate dopamine and glutamate. Their agonists are antiarrhythmic and sedative. Antagonists like theophylline and caffeine are thus stimulants. 2B & 3 involved in immune response.

GASOTRANSMITTERS include carbon dioxide/monoxide, nitric/nitrous oxide and hydrogen sulfide etal.

CARBON DIOXIDE is monitored by the carotid body. High CO_2 will prompt vasodilation to increase blood flow to the area. (carotid body monitors general CO_2 level while other mechanisms regulate local perf.-usion). $CO_2 \rightarrow$ bicarbonate modulates blood pH. Hyperventilation may $\propto$ respiratory alkalosis. Breathing that is too slow or shallow may cause resp. acidosis. A blood test will generally have a reference range of between 20-30 mmol/L. Excessive blood CO_2 is called hypercarbia or hypercapnia, I may be prompted by COPD, sleep apnea, alpha-1-antitrypsin deficiency, muscu. dystrophy, ALS (Lou Gehrig's disease), Guillain-Barre, brain swelling/enceohal-litis & others. Symptoms which require medical attention include confusion, depression, paranoia, compensatory hyperventilation, arrhythmia, panic attack, seizures, muscle twitching and even coma.

NITRIC OXIDE is present and dynamic in almost all types of organisms. Aka "endothelium-derived relaxing factor." Endogenously it is produced from ARGININE, oxygen and NADPH; nitrates may also be reduced to produce it, with nitrite being the intermediate compound. Spinach, arugula, and beets etc. high in nitrate, and have been shown to ↑ NO, with mouth bacteria doing the reducing. NO is a strong vasodilator. Those living in high altitude will see ↑ NO levels as the body attempts to check hypoxia. Nitroglycerin and amyl nitrate ("poppers") are both converted to it. Sildenafil/Viagra works because of it. It is also involved in the hair cycle and minoxidil stimulates it. Phagocytes of the immune system, to kill bacteria and parasites, release it as a free radical. However excessive NO release can promote oxidative damage due to peroxynitrite. NO ↓s the heme in hemoglobin. NO is a calcium channel opener, by this it relaxes smooth muscle. NO action may be impaired in diabetes, atherosclerosis, and ↑ bp.



NITROUS OXIDE is the reductionproductof NO, most often in the liver but it may also be produced by some bacteria. It can alter hemoglobin though through a different process than NO. Aka "laughing gas" it modulates opioid receptors as well as inhibits NMD-Aspartate activity (see both elsewhere in CHEM). It ↑s homocysteine & ↓s methionine.

CARBON MONOXIDE is well known to be toxic when inhaled but surprisingly it is also produced naturally by the human body. It relaxes blood vessels and inhibits platelet clumping. In mammals it is produced from the breakdown of heme. Defective metabolism of it implicated in ↑bp, heart failure, & inflammation.

HYDROGEN SULFIDE is a blood vessel relaxer like NO but it seems to prefer small vessels while NO prefers large; studies suggest they are cooperative. H_2S is also, like NO, involved in penile erection. H_2S supplementation after myocardial infarction may reduce its complications and promote blood flow. H_2S concentration has been found to be severely decreased in Alzheimer's and Parkinson's. In Down syndrome the body produces an excess of H_2S. In Type 1 diabetes an excess of pancreatic beta cells leads to their death and ↓ insulin. Curiously, mice whose air supply was infused with a low dosage of H_2S had their breath rate lowered from 120 bpm to 10 and they were able to survive a 35° drop in body temperature for 6 hrs with no adverse after-effects. It may therefore hold promise in the future for human "suspended animation." It also is responsible for some of the aroma of flatuence. Back to non-gas.

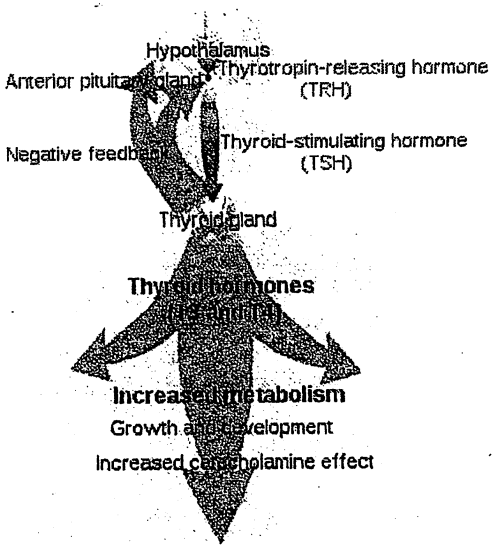
BOMBESIN is aka Neuromedin B (not to be confused with Neuromedin or Neuropeotide B, both presented elsewhere here). After choleocystkinin, it is the second-major neurosignaller to stop eating behavior. It may also be a marker of some lung, gastric pancreatic and nerve cancer. It stimulates gastrin release and may have a role in social behavior.

MELANOCYTE-STIMULATING HORMONE is produced in the pituitary. Its progenitor also produces ACTH (see ADRENALS earlier). It is secreted in response to UV light and thereby increases melanin, but it may also be secreted by the hypoth. when prompted by LEPTIN. Secretion there suppresses appetite as well as sexual arousal & erection (when received by MC3/4 receptors). Many redheads and other fair-skinned people's receptors don't properly respond to it.

AGOUTI-RELATED PEPTIDE is co-expressed with Neuropeptide Y in the hypoth but is active also in the adrenals as well as testes, lungs & kidneys. It is a potent appetite stimulator (opposing MSH). Unsurprisingly it is ↑ by GHRELIN, ↓ by LEPTIN. It is released during fasting and ratchets down TRH (see THYROXINE) to conserve energy. Injection into mice prompted overeating and obesity.

MELANIN-CONCENTRATING HORMONE is another hypothalamic transmitter involved in sleep/wake, depression/anxiety, anorexia, reproduction, maternal behavior, and, not surprisingly, some forms of skin cancer.

MOTILIN is chemically similar to Ghrelin. It, dependent upon pH, prompts GI tract contraction & motility. Glucose (via insulin) ↓s it while erythromycin ↑s it. Motilin in turn increases release of SOMATOSTATIN from the pancreas & small intestine. SS acts as a digestive "brake," inhibiting stomach acid production as well as gastrin, again like motilin, depending on pH (low promotes it, high motilin). SS expressed by the hypoth, received by the pituitary, inhibits growth. Since its neurons also may be found in the hippoc. & solitary tract, it may have other neuroeffects.



THYROXINE (T4) & TRIIODOTHYRONINE (T3) are produced in the thyroid gland by 3 or 4 atoms of iodine combined w/ tyrosine. The hypothalamus releases TRH which is received by the pituitary which then in turn releases TSH. This received by the thyroid prompts release of T4 & T3. When they are elevated feedback ↓s TSH. Likewise when TSH elevated feedback ↓s TRH. Also cold may ↑ TRH to stimulate thermogenesis. However dopamine, SOMATOSTATIN and glucocorticoids (see ADRENALS) may ↓ TSH production.

The primary effect of the Ts is to regulate the metabolic rate but they do much more. In the gut they ↑ absorption of nutrients and the breakdown of fats and glucose. Thus they increase free fatty acids but paradoxically decrease cholesterol levels (by increasing it in bile). They ↑ breath & heart rates. They increase appetite as well as thought generation but they decrease focus. Libido and normal menstruation also modulated by the Ts.

Hyperthyroidism may be caused by Graves disease, goiter, solitary thyroid adenoma or by inflammation. Symptoms include ↑ appetite, but ↓ weight, insomnia, tremor, palpitations, heat intolerance, anxiety, hair loss, diarrhea, chest pain and muscle weakness. (See CHALLENGES section for PARATHYROID.)

A goiter maybe present in both hyper- & hypothyroidism. 5% of the population will develop one. Some are painful but many are asymptomatic. They (and thyroid cancers) are "euthyroid" if no abnormal hormone level found.

CALCITONIN is also produced by the thyroid. In response to high blood calcium it is released, thus ↓ing bone calcium release. This is opposite the effect of PARATHYROID HORMONE (see CHALLENGES). However the latter is more significant as calcium metabolism remains normal after removal of the thyroid.

CALCITONIN GENE-RELATED PEPTIDE is ratcheted down by an increase in intracellular calcium. However, CGRP is much more involv-involved in pain signalling and vascular tone than in Ca balance. CGRP receptors are found throughout the body, indicating its role in respiration, GI function, immune response and gland secretion. It is a vasodilator but is found to be elevated in high bp. Its levels in blood also rise during a migraine, being released from the trigeminal nerve and and the spinal cord. Several drugs have been recently introduced which block its action, including Erenumab/Aimovig, Fremanezab/Ajovy and Galcanezumab. All of these are by injection only. Besides promoting nociception, though there's also evidence it helps heal injured nerves

PHENOTHIAZINES are mood stabilizers that also quell nausea. Included are Prochlorpemazine/Compazine, Stelazine, Promethazine/Phenergan, Prolixin/Fluphenazine, Thorazine and Mellaril.

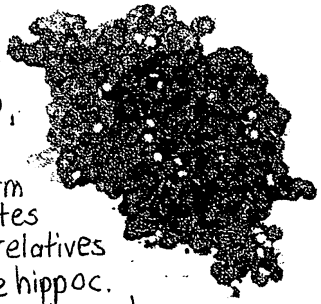
CANNABINOID RECEPTORS- The effect of THC on these is well-known but it is not the only binder of them. ANANDAMIDE (from the Sanskrit Ananda, "Bliss," also Buddha's foremost disciple) also binds them. Endogenously it is produced from lecithin and cephalin, with the omega-6 PFA arachidonic acid as the intermediate compound. In the diet, arachidonic and other fatty acids can be consumed to ↑ anandamide levels. Significantly it and its mimics N-OEA & N-LOEA are to be found in chocolate. Much of what is known about it comes from rodent studies. In them it impairs working memory but increases appetite. Interestingly, it may have been conserved in evolution more due to its fertility aspect than psychoactive effects: it is important in the female for early embryo implantation in the uterus. Its levels meta with gonadotrophin and estradiol. But its psychoactive qualities, in mice at least, correlate w/ THC as it enhances pleasure/reward when injected into their basal ganglia nucleus accumbens. It also in rodents may produce obesity. Some surprising compounds ↑ anandamide, including black pepper and acetaminophen, which may partially explain its analgesia. A case study of a woman who could not produce the enzyme which inactivates it showed she could not feel pain and also was immune to fear and anxiety. Her frequent burns and cuts healed faster than average.

NOCICEPTIN is the first example of reverse pharmacology: its receptor was discovered before it (1995). It is related to the endopioids (see later in CHEM) but does not bind any of them rather it binds the NOP receptor. However such binding does indirectly ↓ the activity of the mu and kappa opioid receptors thus it has been studied as a partial antiopiate. NOP opens potassium channels but blocks calcium ones. Nociceptin and NOP are found throughout the brain and spinal cord. When administered directly to the spine it reduces pain like codeine etc. But when administered in the brain it ↑s pain perception, thus enhancing its antiaddictive effect. It may inhibit GABA and block the dopamine transporter. In animal studies it dampens subjects resampling of meth, cocaine and alcohol, besides the opioids. It has been shown to also be involved in memory, cardiovascular & renal function, locomotor activity, and GI motility.

Anandamide is produced from PHOSPHATIDYLETHANOLAMINE (PE) which itself has been produced from PHOSPHATIDYLCHOLINE (PC) and CEPHALIN, modulated by bile acid. Cephalin is found in ejaculate and of course elsewhere. Both PE and PC are phospholipids and, converse to the above, PC can be produced from PE by action of methionine. All living cells contain PE but it is particularly found in brain white matter, nerves, & the spinal cord. When cardiac blood flow is restricted, PE's control of cell membranes there is disrupted. It and PC also ↑ thrombin and thus regulate blood clotting. PE has a unique property in that it requires no assistance to propagate prions (the agent of Mad Cow disease, et al.) In digested food PE (high in chocolate, soy, egg yolk etc) breaks down to Amadori products, which are implicated in diabetes & cancers associated with it. Both PE & PC are major components of LECITHIN. PC has been claimed to be a treatment for dementia and a lipolytic alternative to liposuction but study results have been mixed. It has been effective through vs Ulcerative colitis

ACETYLCHOLINE (Ach) is received by muscarinic and nicotinic receptors. The ACE enzyme degrades it thus an ACE inhibitor makes Ach more available. They include Donazepril/Aricept, Rivastigmine/Evelon, Galantamine/Rozadyne/Remenyl. They improve divided more than selected attention, and they improve memory but can prompt aggression. Direct Ach ↑ers include physostigmine and tacrine but are not as often used as ACE inhibitors due to their pronounced side effects. Anticholinergics are found amongst many medicine classes; for instance, most antihistamines are anticholinergic (but Tetracyclic Antidepressants are not generally) Ach blockers include Scopalamine as well as the antidizziness medication Antivert.

BRAIN-DERIVED NEUROTROPHIC FACTOR



Without it, the parts of the brain able to grow new neurons (neuroplasticity) could not. Increased by niacin, certain types of exercise, calcium influx via cellular channels, curcumin, calorie restriction, some antideoressants, glutamate, D5 reception in the PFC, and environment/intellectual enrichment. When received by the enzyme Trkb it aids short term memory but the receptor p75 binding it promotes apoptosis rather than growth. (p75 also binds its relatives neurotrophin and nerve growth factor). Besides the hippoc. and cortex, BDNF found in retina, kidney, prostate, saliva and skeletal muscle. It may regulate some nicotinic receptors. It is defective in Alzheimers, Parkinsons, and anxiety disorder. It is ↓ in schizophrenia and implicated in depression, anorexia/bulimia, OCD, and Huntington's. "Brain fatigue" may be due to its interaction with IL-6. Corticosteroids ↓ it (thus observed hippoc. shrinkage). It decreases with age. In the VTA it produces an opiate-like reward state. It is also ↑ in epilepsy. Short-term it ↑s pain sensitivity but long-term it blunts it. It is opposed by GABA.

NEUROKIN/NS K, B & A - A is also SUBSTANCE P when A is in its long form. SubP involved in vomiting, its blockers are the antiemetic PITANTS. NKA blockers are antidepressant while NKB blockers appear to be antipsychotic. All NKs are vasodilators. NK blockers are being studied as treatments for asthma and Irritable Bowel Syndrome.

PINOLENE - an endogenous monoamine oxidase inhibitor. Some believe it to be a co-product of melatonin metabolism. Even at trace concentrations it promotes neurogenesis (in vitro). It is an antioxidant and was shown more effective than Vit. E in retinal damage.

OXYTOCIN is produced in the hypoth, released by the pituitary, and received by the amygdala, where it inhibits fear. Its release stimulated by uterine stretching during labor and nipple sucking. Estrogen ↑s it while (in mice at least) testosterone ↓s it. It suppresses appetite. Often called "the love hormone," it ↑s pair-bonding behavior. Paradoxically, however, also found to ↑ envy and even ↑ Schadenfraude. Sildenafil/Viagra may ↑ it as may 5HT1A ↑ers like Buspar. Possibly defective in autism.

MAGNESIUM - Antagonizes Ca^{2} thus it enhances CaC blocker action. Taken as a supplement (such as Milk of -) it is a mild sedative at doses below that which loosens the bowels (see also BRAIN SAND in CHALL.)

OREXIN Two types, two receptors. A binds to OX1 & OX2 receptors equally but B prefers OX2. Without orexin "brown fat" can't develop thus its thermogenesis inhibited. OXA paradoxically increases lipid storage and ↓s lipolysis. It is theorized then that mice without it grow morbidly obese due to non-OXA factors increasing white fat at the expense of brown. LEPTIN produced by white fat inhibits OX production as does glucose (hyperglycemia also ↓s OX2). GHRELIN as well as amino acids can activate it. "Craving" OXergic neurons are found in PFC, insula, and VTA thus it's involved in addiction. Also active in the hypoth. prompting "wake and rise." Thus narcolepsy more often than not is lack of it. Specifically, narcoleptics have mutant HLA complex / T cell receptors which attack OXergic neurons, an autoimmune process. OX neurons transplanted into pons of narcoleptic rats deterred their excessive sleep. Receptors that prompt OX production include glutamate, cannabinoid, ACH muscarinic-3, 5HT1A, neuropeptide Y, adenosine A1, and cholecystokinin A (below). OX neurons themselves help regulate 5-HT, norepi & ACH.

ENDOTHELIN - 3 forms of it & 4 ET receptors. Platelets binding to LOX-1 receptor causes its release. It is the most powerful vasoconstrictor known however, when binding ET-B, NO2 (see below) is released prompting vasodilation. In insulin resistance, ET-1 activity ↑s thus bp ↑s. ETs implicated in heart failure, cerebral vasospasm, and some cancer's growth. It is involved in pain mediation and maybe a convulsive agent. ET-1 is ↑ in response to hypoxia, inflammation and bacterial toxins. Also received by pituitary.

CHOLECYSTOKININ - Produced in the upper small intestine, is of similar structure as GASTRIN. Released in response to fatty/amino acids mainly but also by a pancreatic peptide CCKRP and ACH from the vagus. It promots bile production & release to digest fat. It suppresses hunger but stimulates the vagus, which may be ↓ed by capsaicin. It cannot cross the BBB but when received by the non BBBed hypoth. causes anxiety and its fragment CCK4 used to test antianxiety medications. Also implicated in PARKINSON's hallucinations. Its A recep. pancreatic, B gastric/neural, which also binds gastrin.

VASOACTIVE INTESTINAL PEPTIDE is produced in gut, pancreas and brain et al. Is circadian-light prompts its production in the hypoth. During daytime it feeds back to produce more of itself. It ↑s prolactin. In the gut, VIP induces sphincter relaxation and inhibits gastric acid ("digestion done, now eliminate"). It is ↑ in IBD (see CHALLEN.) and may prompt diarrhea. It is a strong vasodilator and is being studied to treat heart failure. It also lubricates the vagina and may strengthen cartilage. When knocked out in certain birds they demonstrated less territorial aggression. It may also ↓ bp and relax the trachea.

AMYLOID PRECURSOR PROTEIN is cleaved into amyloid plaque, with the participation of PRESENILIN. This is an integral part of the pathology of Alzheimers (see CHALLENGES) but amyloidosis may occur in the brain w/o Alz, resulting in ANGIOPATHY, ("brain bleed"). Ingestion of aluminum investigated as a cause. There is currently no cure. (CYSTATIN may also cause Angiopathy also.

HUMAN TRACE AMINO ASSOC RECEPTOR- HTAAR1 is pervasive in the brain and also in pancreatic beta cells. It regulates body weight, hunger, and some cognitive processes. 2 prompts WBC movement and in @15% of persons of Asian heritage it doesn't function. HTAARS 5 6 8 & 9 are poorly studied. Non-1s seem to primarily function as pheremone detectors. Binding to 1 or 2 and sometimes both are aminessuch as tyramine and metabolized dopamine, but also LSD & meth etc.

KISSPEPTIN is the initiator of puberty by prompting GnRH production in the hypoth. to activate the Pituitary. Suppresses melanoma and breast cancer metastasis. Same family as Neuropeptide FF.

GHRELIN is produced in the GI tract when the stomach's empty until gastric stretching halts it. Received by the hypoth, it increases hunger, stomach acid and GI motility. It can pass the BBB so may be exogenously administered. Its receptor functions like the D2 receptor so it plays a role in the VTA reward/addiction process; it also interacts with ACH.

LEPTIN is found on the same cells, its effects opposite of ghrelin's.

GALANIN in the brain is highest in the hypoth. and in the GI tract in the duodenum. Its receptors are in the CNS, pancreas and some tumors. 200% upregulated in Alzheimer's (late stage), hypothesized to be a last ditch neuroprotective response though it worsens cognition by ACH being decreased. Opp. glutamate, but not GABA, in the hippoc. So can be anticonvuls ant. Animals with a defective version exhibit ↓ Substance P and nerve injury healing. Also implicated in wake/sleep cycling, feeding, pain & bp regulation.

NEUROMEDIN-U - 2 receptors. NMUR1 is peripheral, mostly in GI tract. 2 is CNS, mostly in medulla, hypoth. and spinal cord. "U" is for uterus where it causes contraction there as well as other smooth muscle (in males too). It ↑s heart rate and plasma epi but not norepi; however, it is also considered a hypoth./symp. NS "brake." It acts via calcium channels. Upregulated in pancreatic cancer. It reduces pain sensitivity.

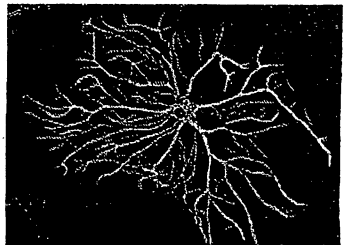
NEUROPEPTIDE-Y is the most abundant peptide in the mammalian CNS. It is a strong vasoconstrictor and causes fat tissue growth. Produced in the hypoth. et al it increases food intake and energy storage, reduces pain perception & anxiety, lowers bp and voluntary alcohol intake and is antiepileptic. It appears to be circadian.

NEUROPEPTIDE-S Mainly produced by amyg. but present elsewhere in brain where it enhances dopamine and reduces anxiety. Animal studies have also shown it to reduce appetite, induce waking, prompt hypersexuality and participate in the extinction of conditioned fear.

NEUROPEPTIDE-B is associated with feeding, memory, learning and the afferent pain pathway. Highest in substantia nigra, hypoth., hippoc. and spinal cord. Related in location and function is NEUROPEPTIDE W.

Other neuropeptides (there are 100+ different types in mammals) include ~~A~~melanocyte Stimulating Horm. and CART-Cocaine and Amphetamine Regulated Transcript. But not enough space!

CHALLENGES - Again, I must be selective due to space. I've mixed "psychological disorders" among "medical diseases" because it's a false and unhelpful distinction. I must first here acknowledge the supreme challenge faced by those who struggle with addiction



Astrocyte

or abuse-not insurmountable but both take an often daily effort to "thrive despite." I take responsibility for the stress and confusion I have brought into the lives of people I loved. Projects like this that I hope are of benefit to others are one way I attempt to atone.

CONVERSION DISORDER/FNSD is probably my primary impetus for producing this zine. Once upon a time women were (ignorantly) diagnosed with hysteria and both genders with hypochondria. Then in the mid 20th century this challenge came to be recognized. Unlike hypochondria, there are actual neurophysical symptoms but they occur without an obvious "nonpsychological" cause. It is diagnosed in 2x to 5x more females than males, but the uterus has long been disproven, of course, to be of itself neuropathological. But CD remains controversial: In the DSM-5 it was reclassified as a type of FUNCTIONAL NEUROLOGICAL SYMPTOM DISORDER. In the DSM-IV it was a somatoform disorder (such as anorexia/bulimia); in the ICD-10 it is rather a dissociative disorder. Common symptoms include blindness, paralysis, deafness, numbness, difficulty swallowing, balance problems, tremors, incontinence, inability to swallow/lump in throat, and fainting. A hallmark of it is the "partial seizure." These are non epileptic and focused on one part of the brain or nervous system, and may have once been called "fits." CD requires a psychological stressor for diagnosis (to "convert" to a symptom), FNSD doesn't. Peak onset seems to be mid 30s. There hasn't been an established cure but hypnosis, EEG biofeedback training, and cognitive behav. ther. etc. may help.

NEURODEGENERATIVE BRAIN IRON ACCUMULATION - While iron is essential, its build up is toxic. Most common in basal ganglia. Symptoms including visual disturbance, movement disruption, seizures, cogn. decline.

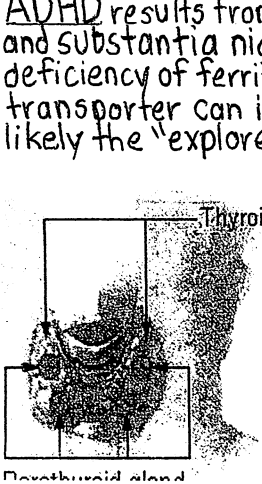
DEPRESSION Marked by atraphy of hippoc. et al. mTOR implicated in. In BIPOLAR amyg. observed to be ↓ in volume but ↑ in activity to PFC.

ANXIETY - Left amyg. hyperactivity linked to separation anx.. Neuralgia, hallucination & delirium observed besides panic. Anxiolytics include GABA ↑ers as well as Buspar/Buspirone and natural products like chamomile.

DELIRIUM can be considered an attention disorder & state between wake/sleep in which ACH ↓. Thus often treated by ACE inhibitors.

In PTSD OCD and BORDERLINE left amyg. has been implicated.

ADHD results from disruption of dopamine projections from the VTA and substantia nigra into basal ganglia. Some studies implicate a deficiency of ferritin but this contraversial (A deficiency of this iron transporter can indubitably cause NBIA (above)). The cause is more likely the "explorer gene." Some therapies are stimulants (Adderall, Ritalin, Vyvanse, Focalin, Concerta & Metadate) some aren't (Strattera). Also used are Wellbutrin, Tofranil, Clonidine & Tenex/Intuniv, etc.



HYPER PARATHYROIDISM - May result from a tumor or anything that prompts ↓ blood calcium or Vit. D deficiency. To correct these the gland overproduces PTH. Hypercalcemia then results, symptomized by fatigue, depression, confusion, GI/head pain, renal issues.

ALZHEIMER'S - Mammillary body atrophy associated with. Defective BDNF is ↑ in Alz patients, this may explain hippocampal shrinkage also observed in Alz. Buildup here is twofold: amyloid beta plaque & tau protein. mTOR is ↑ in Alz initially. Amyloid plaque shown to activate mTOR until the plaques become large & cytotoxic then mTOR ↓. PRAS-40 found to be significant cause of Alz cognitive impairment. PRAS-40 is actually a component of mTOR. eIF-2 is a downstream target of mTOR. eIF-2 shown to be a mediator of cell death in prion diseases (like mad cow disease) which Alz resembles in its tau protein nerve fiber tangling, which mTOR promotes. Specific loss of episodic memory here indicates right amygdala damage. (see also AMYLOID PRECURSOR in CHEMISTRY)

DYSAUTONOMIA Signals sent by the autonomic n.s. to the organs are either insufficient or unbalanced between the symp. and the parasymp.. Numerous causes include alcoholism, amyloidosis, lupus, neck injury, AIDS, diabetes, Lyme disease, multiple sclerosis, Parkinsons, neurosurgery etc. Clustered often with fibromyalgia, chronic fatigue, interstital cystitis and IBS; it could be the underlying cause of all these. Anxiety may also be a cause of it as well as a symptom. Other symptoms (which are numerous) include blurry/double vision, bowel incontience/constipation, brain fog, dizziness, difficulty swallowing, insomnia, ↓ bp (which may be orthostatic), fainting, ↑ heart rate, urinary incontinence/retention and weakness. Testing for it includes cardiopulmonary performance, adrenergic response and thermoregulatory sweat assay (another symptom of it can be anhydrosis - ↓ sweating). Also used is the Valsalva maneuver pictured above. When one normally exhales with force while pinching the nose closed and with the mouth closed, heart stroke volume rises. Between 5-14 seconds into it bp compensates and stroke volume falls. The resulting vasoconstriction then begins to normalize bp until is status quo at @ 30 seconds. Meanwhile the pulse rate has reflexively peaked at 20 seconds and then also normalized by 30. Valsalva is used also to diagnose vertebral injury because pressure in the spine rises during it making pain more acute. Headache when performing Valsalva one of main symptoms of ARNOLD-CHIARI malformation associated with some migraines. Valsalva can also make neck lymph nodes more prominent which can then be palpitated for cancer diagnosis (well over half of malignant cancers show lymph node swelling there). Valsalva is also used to prompt defecation - but caution! as this has been linked to heart attack.



SCHIZOPHRENIA - Mammillary body atrophy associated with, as well as ↓ BDNF in the DLPFC. Appears to result from disruption of dopaminergic neurons projecting from the VTA and substantia nigra to the basal ganglia. Many consider SCHIZOAFFECTIVE and -TYPAL to be milder forms on a continuum rather than distinct entities. Among many treatments are Paliperidone/Invega, Abilify/Aripazole, Geodon/Ziprazidone, Haloperidol, Lurasidone/Latuda, Olanzipine/Zyprexa, et al.

MIGRAINE Essentially a travelling cerebral vasoconstriction in response to a disrupting stimulus which is often idiosyncratic but often includes sleep cycle disruption, barometric pressure, strobe lights, caffeine, aspartame, tyramine etc. The CTZ is actived thus nausea. Pain, often focused to one hemisphere, is considered the reactive vasodilation. In Classical, an aura occurs prior to pain, the result of altered blood flow/biochemistry of sensory neurons.
ABDOMINAL MIGRAINES, more recently recognized, are probably the same challenge as CYCLICAL VOMITING SYNDROME.
Because migraines demonstrate ↓ 5-HT, triptans are a treatment as are propanalol, certain antidepressants, and antihistamines like Pizotifen as well as some CaC blockers like Lamotigrine.
IRRITABLE BOWEL SYNDROME/INFLAMMATORY BOWEL DIS.
In IBD there is an obvious inflammatory source: Crohn's is autoimmune source. IBD may display non-bowel symptoms such as uveitis & iritis (both of the eye). IBS has a recognized neurological componement and may be treated w/ anticholinergics and serotonin modulators such as Alosetron and Cilansetron. VIP also implicated here.



PHEOCHROMOCYTOMA Tumor arising from catecholamine secreting cells, usually norepi, occasionally epi, and rarely dopamine. Usually an adrenal mass but can also arise in enterochromaffin-like (ECL) cells in the gut & elsewhere, where they are called paraganglioma. Cantinuous or intermittent catecholamine secretion causes bp swings, headaches, palpitations, sweating and night terrors. Some have been successfully treated with nonselective alpha blockers.
CORPOREA ARENACEA/BRAIN SAND Common deposition of calcium magnesium and ammonium phosphate in the aging brain. Most often observed in the pineal.
PORENCEPHALY - Brain tissue cyst that is not a benign or malignant tumor, or a cavity that is not normal in the ventricles. Colloid cysts in the 3rd ventricle associated with atrophied mammillary bodies.
ENCEPHALOMALMACIA - Brain tissue loss/softening.
PARKINSON'S Hippoc. shrinkage (associated w/ defective BDNF) correlated with. Appears to result from disruption of dopaminergic projections from the substantia nigra and the VTA to the Basal Ganglia thus D is depleted there. mTOR found to be ↑ in. It is a common cause of DYSAUTONOMIA and results in a poor prognosis for those patients. Choleocystokinin implicated in its hallucinations
AKATHISIA may be a sign of Parkinson's but also caused by D receptor blockers, some SSRIs, metoclopramide, reserpine and untreated schizophrenia. However, half of antipsychotic users will also develop it. Symptoms range from fidgeting, rocking, & pacing to pain & terror. Benadryl, trazodone, Remeron, Beta blockers, iron and Vit. B can treat it.

NEUROLEPTIC MALIGNANT SYNDROME is rare (1 of 5000 or so) but demonstrates the elevated care that those on antipsychotic and other neuroactive medicine should receive. Symptoms include muscle cramps/rigidity/tremor, fever, ↑WBC count, sweating, bp swings, and delirium which can progress to coma. 10% fatal. There may be a genetic predisposition and postpartum women and Lewy body dementia patients are at greater risk. A complex combination of factors implicated in its origin (altered dopamine reception, ↑5-HT-1 reception, ↑glutamine, ↓GABA, ↑sympathetic activity and muscle cell calcium release). The effects of meth and cocaine may be mistaken for it. Many think it to be a drug-induced form of malignant hypertension/catatonia. Treat w/ dopamine ↑ers/D2 activators/sympathetic ↓ers.

SEROTONIN SYNDROME resembles NMS but is more common. Here there is an excess of 5-HT with symptoms like the above though without muscle rigidity or the ↑white blood cells of NMS.

ANTIDEPRESSANT WITHDRAWAL SYNDROME- 20-50% of people who abruptly stop taking a SSRI, SNRI, MAOI, or tricyclic antidepressant experience this. Symptoms vary but include insomnia, nausea, poor balance, anxiety, flu-like symptoms, sensory disturbance, tremors, and, rarely, psychosis. Common are electric-shock like experiences called brain zaps or brain shivers. Paroxetine/Paxil, Venlafaxine/Effexor, and Duloxetine/Cymbalta are considered the worst offenders. Fluoxetine/Prozac may be considered the least likely to cause due to its long action. The exact mechanism of AWS is unknown but it parallels withdrawal from known addictive psychotropics like benzodiazepines. PAWS (Prolonged AWS) is over 18 months, but usually AWS resolves within a few weeks.

APHASIA - Speech/language loss. Fluent-Temporal Wernicke's is damaged, can't comprehend oral/written. Nanfluent-OVPC Broca's damaged, can't organize language in order to verbalize. Conduction-damage to Arcuate Fasciolus that connects Wernicke's and Broca's-can't reproduce heard speech. Anomic-dam. to corpus callosum.

SEMANTIC DEMENTIA - Rare. Language loses meaning while speech itself as well as nonverbal problem solving and episodic memory preserved. Associated w/damage to ventral nucleus and mammillary bodies.

ECZEMA High levels of BDNF and Substance P associated with Increased itching.

SLEEP APNEA Associated with atrophied mammillary bodies.

CONGESTIVE HEART FAILURE - Also associated w/ atrophied mammillary bodies. Symptoms may be mitigated by some alpha and Beta blockers, promoting vasodilation and ↓ cardiac output.

ALCOHOLIC KORSAKOFF SYND. Malnutritional thiamine (B1) deficiency. Dementia/amnesia due to PFC/limbus/cerebellum damage.

CLOSING THOUGHTS... (or further opening?)

Though I claim affinity with Taoism/Humanism/Naturalism, inmates generally must identify one religious affiliation "for the system," so mine is Buddhism and I am most copacetic with Zen. However, I've struggled with one of its central facets: no-mind, which is also expressed somewhat clandestinely in this zinelet's title. In Buddhism: Plain & Simple Steve Hagen quotes Master Katagiri Roshi: (We need) "to rest our frontal lobes." A scan of even the brief info presented here, though, reveals that such a state-"hypofrontality" exists in many of what I've termed brain challenges where there is no check on what McLean in 1952 coined "the Visceral Brain"-the amygdala, etc. And of course w/o frontal lobes, no koans of Zen would ever be recorded; there would be no history with context, no ideation to foster new Truth nor process to get at its meaning (philosophy). So Zen is right to claim all is flux and that it's a trap to cleave the impermanent mind from the rest of impermanent matter-energy but "I"-whatever that means- think the baby need not be thrown out with the (muddy) water. Emotions - the language of the cerebral limbus- need not be considered evolutionary artifacts but rather are for the most part naturally selected benefits. No fear, and our ancestors don't run from life-threats and the species perishes before its "higher thought" emerges. Without sadness, could there be empathy for another's pain? Rage & worry (problems of my crisis) may be a bit more difficult to accomodate. Yet without worry (unless it's paralyzing) is "If I don't, who will?" ever asked and doesn't anger at the patently wrong prompt action to right it? Mirth speaks for itself or just smiles. And with all these the body is more copilot than passenger, the testimony of this zine. So balance is ultimately key. When one learns (or, some teach, recovers) how to feel the body talking to the mind & changing it, to know too what homeostasis feels like and when the chemistry's out of whack (as it may have been for a lifetime), then one is able reclaim a measure of care for oneself (that is what prison is all about removing in the first place). I wish you success in taking back what's been yours all along, even if it is, as this guy ← elucidated, inherently impermanent!



GOLF

One of those simple ones that you always seem to lose by one or two. 7 rows of 5 FD. Turn FU all at right end of rows. One of these moved out of layout starts your Putt Pile. Take end card from row onto putt pile if it's one lower or higher in rank, suit doesn't matter. When stuck, turn over one from stock of 17 and continue up/down on it. When FU end of row moved to Putt, turn new end of row FU. Your tee time is over w/ a LOSS if you don't end up w/ just Putt P.



LOUISE MICHEL

WINGS/13 DOWN

Deal 13 down in middle, this is the breast. Now go 4 FU on l&r sides of breast, the wings. 1 more goes FU on top, this is one of the Foundations. Other 3 of that same rank go up there as they are revealed. Turn top of breast over. This card and wings played to Foundations, building up in suit. Stock is the bird's tailfeather, keep turning over its top card to go to wings gaps or directly to Foundation. If it can't, FU in waste. Top of waste can go up too. If you go thru stock/waste w/o any new plays, a loss. Also called Eagle's Wings for the (ugh) patriatic... in same vein...

BETSY ROSS/MUSICAL/FAIREST/QUADRUPLE ALLIANCE/PLUS BELLE/4 KINGS Lay out any 2 3 4 A, suits don't matter. Below them 4 6 8 2. Top to remind you of increments. On 2s build by 1s up to K. On 4 up by 2s, on 6 by 3s, on 8 by 4s (8→Q→3→7→J→2→6→10→A→5→9→K), 2 redeals. Both of these are of moderate difficulty.

BARONESS/13/5 PILES

5 FU. Remove K or any pair that sums 13 (A+Q, J+2 etc) Discard those. Next 5 on top of those. Discard all to win, which is infrequent. Yes, Ms. Parsons, I do regret all these elitist titles - but at least the brass can be conquered (for a change?) here!



LUCY PARSONS

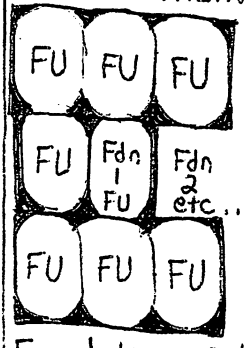


12 anarchists who changed the world

CHE

DOUBLER/QUITS

7 FU, 8th also FU is 1st Foundation:



Foundations 2-4 FU of same rank as #1, as revealed. From stock or walls build Fdns following this (Doubler) pattern: 2→4→8→3→6→Q→J→9→5→10→7→A→2. Fill in wall space from stock or waste top. 4 completed Fdns wins!



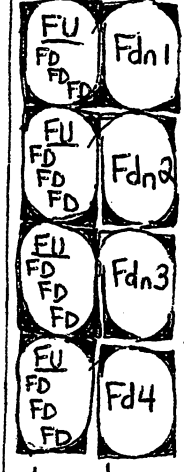
JOHN LENNON

DUCHESS/GLENWOOD

(Yeah I know, Commandante, another entitled title. Let us show them revolutionaries play cards too.!) 4 fans of 3 FU above. A space below these for Foundations, then a row of 4 FU cards below that space. Any of the topmost cards on fans may become a Foundation which will build up in suit. It is very helpful to place a Foundation in each suit. Descend in alternating colors from the cards in the row under Foundations by rank. All cards of one of these descents may be moved onto another descent in proper sequence. A card from stock that can't go to Found, or to Descent, goes to Waste, top of which also available. Fill spaces in Desc. from fans, if no fans then from wastepile. The rules for this says one redeal but that seems overly stringent. Go thru waste-turned restock again without a play, you've lost.

OSMOSIS/TREASURE TROVE

4 sets of 4 cards stacked FD, place these in column. Next card in deck is a Foundation. Others of same rank become Foundations as revealed. Turn up top of each column or stack. These will go in the row leading right in suit of the correct Fdn, turn next up. However, if row 1 is ♠ and ♡s go in row 2, a ♡ can only go there after same rank ♠ has gone in top row. If, then, ◇ is row 3, only ◇s that have had their ranks played in ♠s & ♡s can go there. Pattern applies to row four. Once a rank has been played on all 4 rows, it may be collapsed onto Foundations. Exhaust waste/restock & all of left column stacks, you win!



THE GAPS FAMILY

No, not that corporate bastion, rather sols featuring "all layout" in which cards are moved when gaps allow. The p/matriarch (?) would be GAPS/SPACES. Deal entire pack FU unstacked 4X13. Remove As, into their spaces go next higher in rank and suit to card on left (ie 3♡ on left, 4♡ goes to its right). Gap after K must be left empty, action blocked there. Win is a row from 2-K in each suit. Redeal by gathering up all except end of row 2s and insuit sequences from low on left to high on right. The version MONTANA's rules end there with two of these redeals, where the gathered cards are strategically placed after shuffling, the limitation being only 2s may go in extreme left. Apparently G/S allows redeals until blocked. Not too difficult to win, but rather long. Obviously shorter is HOUSE OF COMMONS in which Faces are removed so the deck is 40 and the layout is 4X10. Aces are then removed and into their gaps go cards just like G/S/M. Just as long as G/S/M but much harder is MAZE. Deal stack FU into 6 rows. 1st and 2nd gets 8 cards each, the other rows get 9. Immediately remove Ks thus 6 gaps. A gap filled by same suit 1 below card to right or 1 above card to left. A gap to the right of Q may be filled by an A of a different suit or by the card 1 lower than the card to the gap's right. A win is A in top left corner, complete in suit sequences to Q, with Q in extreme right corner. One redeal only in HoC, none in Maze, sorry!



CESAR CHAVEZ

HIT OR MISS/TRIEZE/HARVEST TALKATIVE/ROLL CALL

This one is good if you have limited or no table space. Actually I won in the middle of an emergency light-only power outage & riot! Say "Ace" as you turn over card #1, take it out if it's an Ace. Next say 2 with card #2 and take it out if it matches etc. If you go 2x thru deck w/o a hit, is a loss. A good cure for boredom if not the disease that Mr. Carpenter called civillization!



EDWARD CARPENTER



RUSSELL MEANS

THE FAMILY OF BAKERS

Of these MARTHA is my favorite but that's only because it is the best named Sol of all time. Any-wha, pull As, they will Found to the side. Then deal 12 columns of 4 each, most recent at the bottom on top of previous, alternating FU & FD. You will build up, As to Ks in suit. In the tableau, descend by alternate color. Groups of cards may be moved as a unit to an available card in another. However-and this the hitch making it less than 100% winnable-a columnar gap may only be filled by a singleton, not a sequence. Still you'll win most of the time, and that's a good thing, right?

BAKER'S DOZEN is more difficult: Deal FU 13 columns of 4, most recent available at bottom, like Martha. A's founded as they become available, will then be built in suit to Ks. The "artificial" part of this one (IMHO) is that Ks that are available at bottom are moved under another in column (4th switches with 3rd, etc.) As well if a K is lying on another of the same suit anywhere (not necessarily available) swap these. Without these swaps this would be an extremely difficult sol, even with, it's considerably harder than Martha. Descent is down-by rank, disregarding color/suit. Don't fill in columnar gaps (the other limiting factor).

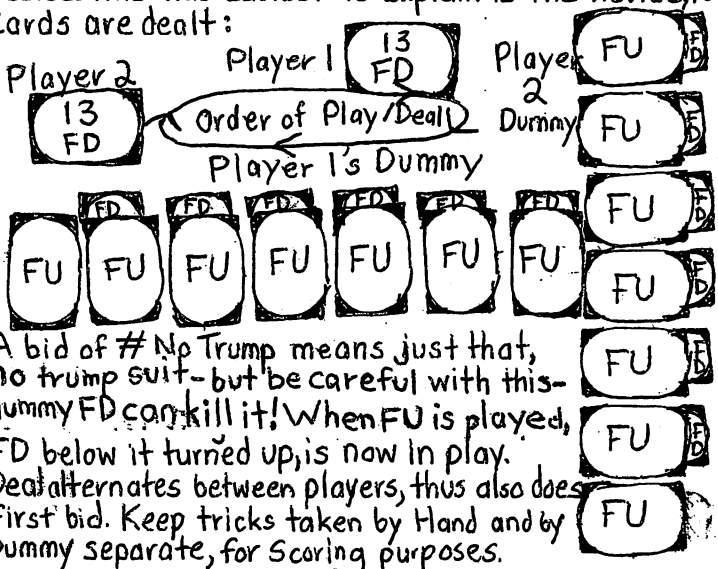


JOSIAH WARREN

If you remove A's at start, B.D. becomes PERSERVERANCE. If you found 2 A's at the start and make your grid 10 columns of 5, it is now GOOD MEASURE. If you don't Found any A's at the start and 2 of your columns have 6 rather than 5 cards, it's called VINEYARD, which is appropriate because we know Martha does like her glass of wine after a long day of baking. (Wine, I stress, not hooch. Patient Activists in detention should avoid that poison-and all others, right?)

HALF-NAKED HONEYMOON AUCTION BRIDGE

(The scoring and mechanics of Contract Bridge (such as "vulnerability") may applied to this instead of the Auction system but I decided this was easiest to explain to the novice). Uses 1 standard deck. Cards are dealt:



Players pick up hands, let Dummies be. Dealer bids 1st, saying # they think they can take (in excess of 6 tricks) if they get to name trump, considering their hand and their dummy's. Suit must be followed. May trump if no suit but don't have to, nor do you have to exceed rank.

A bid of # No Trump means just that, no trump suit - but be careful with this - dummy FD can kill it! When FU is played, FD below it turned up, is now in play. Deal alternates between players, thus also does first bid. Keep tricks taken by Hand and by Dummy separate, for Scoring purposes.

A bid is higher if it contracts for a higher # of bid pts which I'll explain in Scoring. But, for example, 3♠ (27 pts) bids higher than 4♣ (24 pts). (However, those familiar with Contract Bridge bidding could use it instead w/o harm).

Doubling means an opponent believes the bidder will fall short and thus opp. trying to get more bonus. Redoubling means bidder is confident they'll make it and thus gets X4 if successful, but if fail gives X4 to opp. A doubled or redoubled contract maybe nullified by bidding a higher single bid which then of course may then be doubled/re- too.

Passing means you can't beat the previous bid. Passer leads. If Player 1, it's from their hand. If Player 2, from Dummy.

If you don't get it confused with Scoring Bid Pts, a Hand Pts tally can help you decide if/how much to bid (acknowledging FD Dummy cards are a mystery at the bid): 4 for each A, 3 K, 2 Q, 1 J. Add both Hand & visible Dummy's. Under 15, pass. 16-20, Try Bid 1. 21-22, Bid 2. 23-25, Bid 2 or 3. 26-28, Bid 4. 29-30, Bid 5. 30+, go 6 or 7 (remembering Opp. leads) If you have more than 7 of a suit, add pts accordingly, that is a "Golden."

Scoring: Divide with a cross: Me / You Below the hor. line will be Bid pts. taken (past 6): NT scores 10, ♠9, ♡8, ◇7, ♣6 for each trick bid & taken, X2 if doubled, X4 if redoubled. Above the hor. line each undertrick (high bidder fell short) gives 50 to Opp. these also subject to double or redouble. The other above the line bonuses are: 50 for making a doubled contract, 100 for redoubled. 50 for (6/7) Small Slam, 100 for (7/7) Grand Slam, and for Honors.

cont. →

Honor cards are Aces, Faces and Tens. If in EITHER your hand or your dummy's, taken tricks at the end score 80 pts above for 4 Trump Honors or 100 for all 5. In NT for all 4 Aces, score 100. If between you and your dummy you have 3 or more Honor cards (in Trump or Aces NT) then score 10 above for EACH of them.

When a player makes 30 pts below, it's a game, line is drawn to start new game and those are now pts above. When a player has won 2 games they get a 250 pt bonus ("rubber") above, and both players' pts. above and below are totalled. Highest total wins overall.

Bridge's history is a bit murky, BTW. "Biritch" is Russian Whist, Euchre being a diplomat. But others think English Whist was played by British soldiers near the Galata Bridge in Istanbul during the Crimean War. Competitive trump-bidding seems to have arisen in India around the turn of the last century.

JUSE

Most popular in Sri Lanka, it appears to have derived from the 19th century English game Muggins. Ace is high & low. Any # can play. Dealer deals to self first. Last card goes to center as first Foundation. As they are revealed, the 3 other cards of that rank will become Foundations as well. Player & dealer overturn top card, in turn, of their stock. If it is (in suit) 1 rank higher than a Foundation, must be priority played there. Next down in priority is 1 rank higher (any suit), than top of opp's "waste pile." Lowest priority is own waste pile. On next turn, top of own waste pile (which has been played FU) must be priority-played before a new card is overturned from draw stack. If at anytime one of these priority plays is missed, "Juse" is called and caller takes one from bottom of his draw stack, adds to opp's. Winner empties both their draw stack and waste pile. -Markthа, 2019.



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