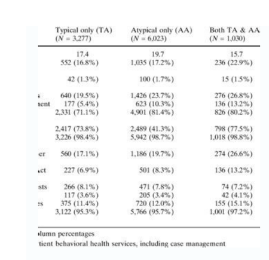
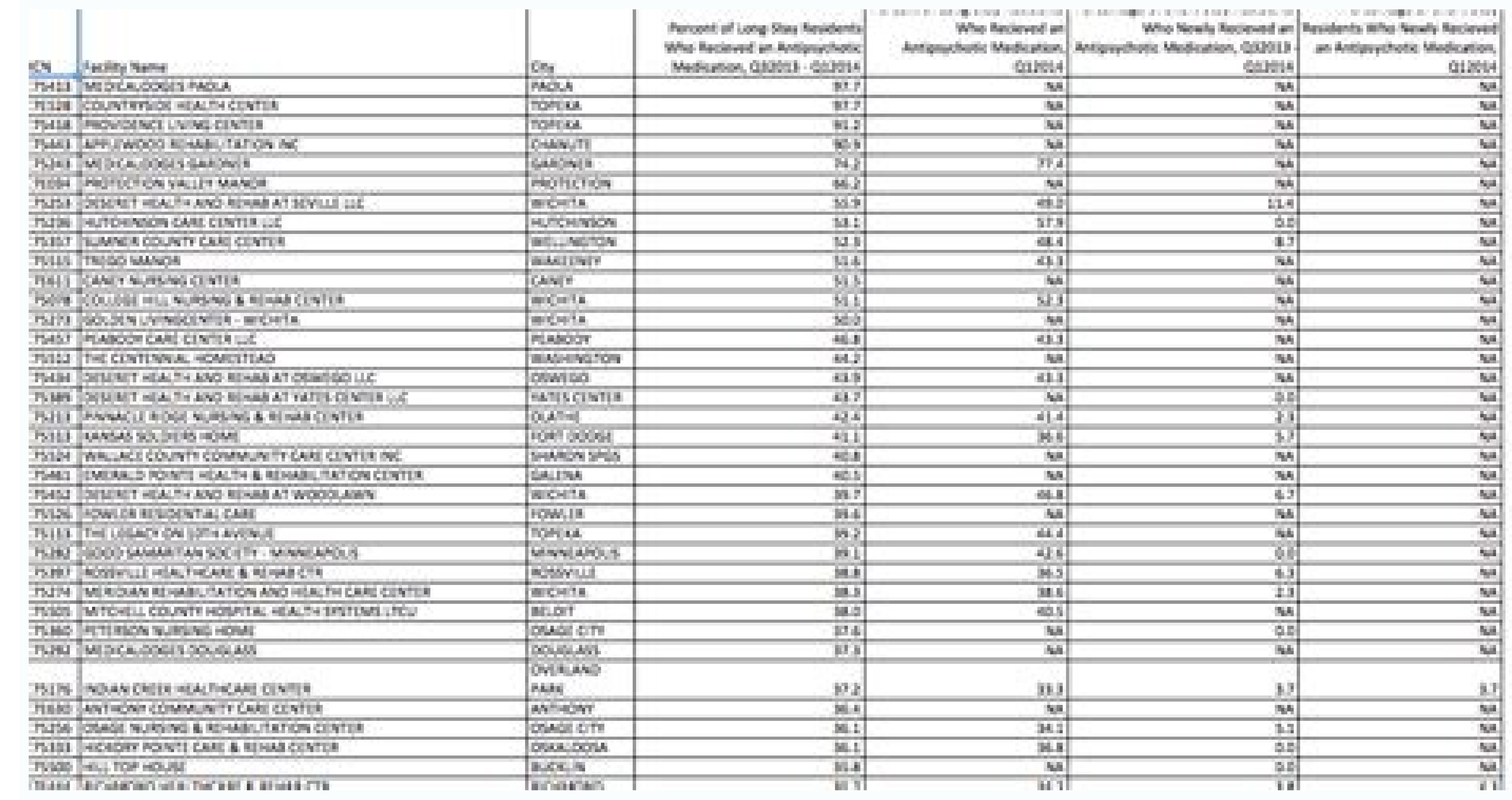
Antipsychotic drug classification pdf

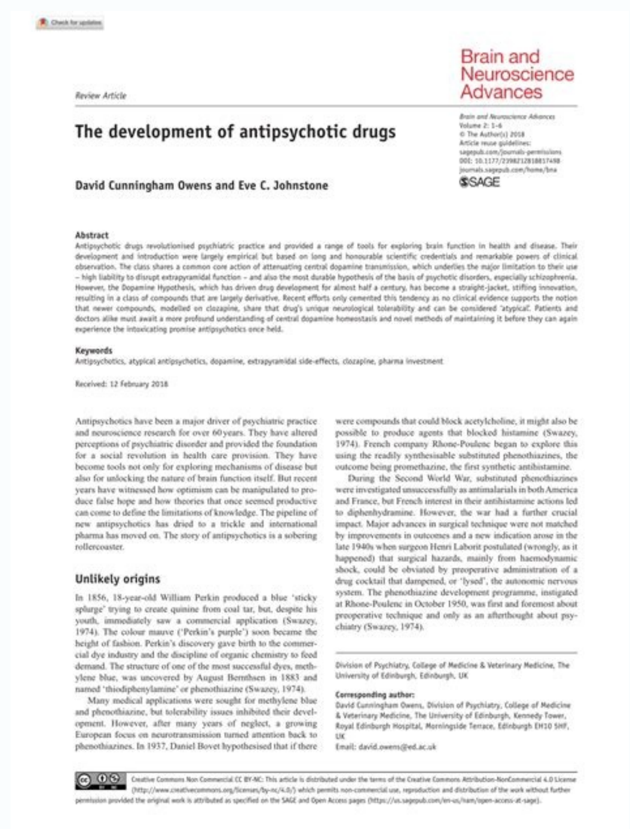


	Typical only (TA) (N = 3,277)	Atypical only (AA) (N = 6,023)	Both TA & AA (N = 1,030)
	17.4	19.7	15.7
	552 (16.8%)	1,035 (17.2%)	236 (22.9%)
	42 (1.3%)	100 (1.7%)	15 (1.5%)
s	640 (19.5%)	1,426 (23.7%)	276 (26.8%)
ient	177 (5.4%)	623 (10.3%)	136 (13.2%)
	2,331 (71.1%)	4,901 (81.4%)	826 (80.2%)
	2,417 (73.8%)	2,489 (41.3%)	798 (77.5%)
	3,226 (98.4%)	5,942 (98.7%)	1,018 (98.8%)
er	560 (17.1%)	1,186 (19.7%)	274 (26.6%)
ict	227 (6.9%)	501 (8.3%)	136 (13.2%)
sts	266 (8.1%)	471 (7.8%)	74 (7.2%)
	117 (3.6%)	205 (3.4%)	42 (4.1%)
rs	375 (11.4%)	720 (12.0%)	155 (15.1%)
	3,122 (95.3%)	5,766 (95.7%)	1,001 (97.2%)

olumn percentages
tient behavioral health services, including case management



ICN	Facility Name	City	Percent of Long-Stay Residents Who Recieved an Antipsychotic Medication, Q32013 - Q12014	Who Recieved an Antipsychotic Medication, Q12014	Who Newly Recieved an Antipsychotic Medication, Q32013 - Q12014	Residents Who Newly Recieved an Antipsychotic Medication, Q12014
75413	MEDICALODGES PAOLA	PAOLA	97.7	NA	NA	NA
75328	COUNTRYSIDE HEALTH CENTER	TOPEKA	97.7	NA	NA	NA
75418	PROVIDENCE LIVING CENTER	TOPEKA	91.2	NA	NA	NA
75443	APPLEWOOD REHABILITATION INC	CHANUTE	90.9	NA	NA	NA
75243	MEDICALODGES GARDNER	GARDNER	74.2	77.4	NA	NA
7E004	PROTECTION VALLEY MANOR	PROTECTION	66.2	NA	NA	NA
75253	DESERET HEALTH AND REHAB AT SEVILLE LLC	WICHITA	55.9	49.0	11.4	NA
75236	HUTCHINSON CARE CENTER LLC	HUTCHINSON	58.1	57.9	0.0	NA
75357	SUMNER COUNTY CARE CENTER	WELLINGTON	52.3	48.4	8.7	NA
75115	TREGO MANOR	WAKEENEY	51.6	43.3	NA	NA
75611	CANEY NURSING CENTER	CANEY	51.5	NA	NA	NA
75078	COLLEGE HILL NURSING & REHAB CENTER	WICHITA	51.1	52.3	NA	NA
75273	GOLDEN LIVINGCENTER - WICHITA	WICHITA	50.0	NA	NA	NA
75457	PEABODY CARE CENTER LLC	PEABODY	46.8	43.3	NA	NA
75512	THE CENTENNIAL HOMESTEAD	WASHINGTON	44.2	NA	NA	NA
75494	DESERET HEALTH AND REHAB AT OSWEGO LLC	OSWEGO	43.9	43.3	NA	NA
75389	DESERET HEALTH AND REHAB AT YATES CENTER LLC	YATES CENTER	43.7	NA	0.0	NA
75213	PINNACLE RIDGE NURSING & REHAB CENTER	OLATHE	42.4	41.4	2.3	NA
75313	KANSAS SOLDIERS HOME	FORT DODGE	41.1	36.6	5.7	NA
75324	WALLACE COUNTY COMMUNITY CARE CENTER INC	SHARON SPGS	40.8	NA	NA	NA
75463	EMERALD POINTE HEALTH & REHABILITATION CENTER	GALENA	40.5	NA	NA	NA
75452	DESERET HEALTH AND REHAB AT WOODLAWN	WICHITA	39.7	46.8	6.7	NA
75526	FOWLER RESIDENTIAL CARE	FOWLER	39.6	NA	NA	NA
75113	THE LEGACY ON 10TH AVENUE	TOPEKA	39.2	44.4	NA	NA
75282	GOOD SAMARITAN SOCIETY - MINNEAPOLIS	MINNEAPOLIS	39.1	42.6	0.0	NA
75397	ROSSVILLE HEALTHCARE & REHAB CTR	ROSSVILLE	38.8	36.5	6.3	NA
75274	MERIDIAN REHABILITATION AND HEALTH CARE CENTER	WICHITA	38.3	38.6	2.3	NA
75305	MITCHELL COUNTY HOSPITAL HEALTH SYSTEMS LTCU	BELOIT	38.0	40.5	NA	NA
75360	PETERSON NURSING HOME	OSAGE CITY	37.6	NA	0.0	NA
75282	MEDICALODGES DOUGLASS	DOUGLASS	37.3	NA	NA	NA
75176	INDIAN CREEK HEALTHCARE CENTER	OVERLAND PARK	37.2	33.3	3.7	3.7
71680	ANTHONY COMMUNITY CARE CENTER	ANTHONY	36.4	NA	NA	NA
75256	OSAGE NURSING & REHABILITATION CENTER	OSAGE CITY	36.1	34.1	5.1	NA
75101	HICKORY POINTE CARE & REHAB CENTER	OSKALOOSA	36.1	36.8	0.0	NA
75500	HILL TOP HOUSE	BUCKLIN	35.8	NA	0.0	NA
[illegible]	RICHMOND HEALTHCARE & REHAB CTR	RICHMOND	[illegible]	[illegible]	[illegible]	[illegible]



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Review Article

Brain and Neuroscience Advances

The development of antipsychotic drugs

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Abstract
Antipsychotic drugs revolutionised psychiatric practice and provided a range of tools for exploring brain function in health and disease. Their development and introduction were largely empirical but based on long and honourable scientific credentials and remarkable powers of clinical observation. The class shares a common core action of attenuating central dopamine transmission, which underlies the major limitation to their use – high liability to disrupt extrapyramidal function – and also the most durable hypothesis of the basis of psychotic disorders, especially schizophrenia. However, the Dopamine Hypothesis, which has driven drug development for almost half a century, has become a straight-jacket, stifling innovation, resulting in a class of compounds that are largely derivative. Recent efforts only cemented this tendency as no clinical evidence supports the notion that newer compounds, modelled on clozapine, share that drug's unique neurological tolerability and can be considered 'atypical'. Patients and doctors alike must await a more profound understanding of central dopamine homeostasis and novel methods of maintaining it before they can again experience the intoxicating promise antipsychotics once held.

Keywords
Antipsychotics, atypical antipsychotics, dopamine, extrapyramidal side-effects, clozapine, pharma investment

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Antipsychotics have been a major driver of psychiatric practice and neuroscience research for over 60 years. They have altered perceptions of psychiatric disorder and provided the foundation for a social revolution in health care provision. They have become tools not only for exploring mechanisms of disease but also for unlocking the nature of brain function itself. But recent years have witnessed how optimism can be manipulated to produce false hope and how theories that once seemed productive can come to define the limitations of knowledge. The pipeline of new antipsychotics has dried to a trickle and international pharma has moved on. The story of antipsychotics is a sobering rollercoaster.

Unlikely origins

In 1856, 18-year-old William Perkin produced a blue 'sticky splurge' trying to create quinine from coal tar, but, despite his youth, immediately saw a commercial application (Swazey, 1974). The colour mauve ('Perkin's purple') soon became the height of fashion. Perkin's discovery gave birth to the commercial dye industry and the discipline of organic chemistry to feed demand. The structure of one of the most successful dyes, methylene blue, was uncovered by August Bernthsen in 1883 and named 'thiodiphenylamine' or phenothiazine (Swazey, 1974).

Many medical applications were sought for methylene blue and phenothiazine, but tolerability issues inhibited their development. However, after many years of neglect, a growing European focus on neurotransmission turned attention back to phenothiazines. In 1937, Daniel Bovet hypothesised that if there were compounds that could block acetylcholine, it might also be possible to produce agents that blocked histamine (Swazey, 1974). French company Rhone-Poulenc began to explore this using the readily synthesisable substituted phenothiazines, the outcome being promethazine, the first synthetic antihistamine.

During the Second World War, substituted phenothiazines were investigated unsuccessfully as antimalarials in both America and France, but French interest in their antihistamine actions led to diphenhydramine. However, the war had a further crucial impact. Major advances in surgical technique were not matched by improvements in outcomes and a new indication arose in the late 1940s when surgeon Henri Laborit postulated (wrongly, as it happened) that surgical hazards, mainly from haemodynamic shock, could be obviated by preoperative administration of a drug cocktail that dampened, or 'lysed', the autonomic nervous system. The phenothiazine development programme, instigated at Rhone-Poulenc in October 1950, was first and foremost about preoperative technique and only as an afterthought about psychiatry (Swazey, 1974).

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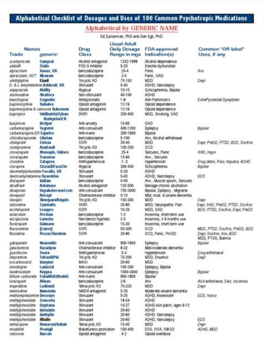
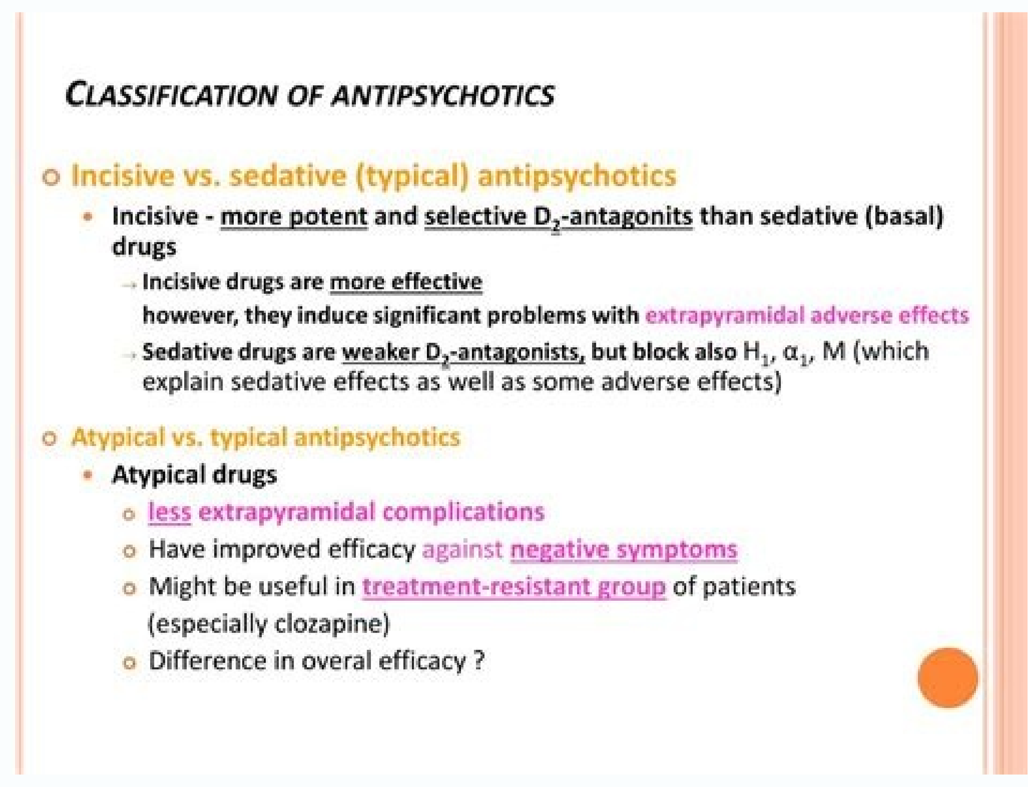


Alphabetical Checklist of Dosages and Uses of 100 Common Psychotropic Medications
Alphabetical by GENERIC NAME



What are the classes of antipsychotics. What are some common antipsychotic drugs. Antipsychotic drug classification pdf. Antipsychotic drugs classification mnemonic. Are the most widely used class of antipsychotic drugs.

About 95% of all antipsychotics prescribed in the US are SGAs. SGAs also do the following: May cause less cognitive blunting Are less likely to have extrapyramidal adverse effects (including a much lower risk of tardive dyskinesia) Increase prolactin slightly or not at all (except risperidone, which increases prolactin as much as do conventional antipsychotics) SGAs may appear to lessen negative symptoms because they are less likely to have parkinsonian adverse effects than conventional antipsychotics. Clozapine, the first SGA, is the only SGA shown to be effective in up to 50% of patients resistant to conventional antipsychotics. Clozapine reduces negative symptoms, reduces suicidality, has few or no motor adverse effects, and has minimal risk of causing tardive dyskinesia, but it has other adverse effects, including sedation, hypotension, tachycardia, weight gain, type 2 diabetes, and increased salivation. It also may cause seizures in a dose-dependent fashion. The most serious adverse effect is agranulocytosis Neutropenia Neutropenia is a reduction in the blood neutrophil count. If it is severe, the risk and severity of bacterial and fungal infections increase. Focal symptoms of infection may be muted, but fever... read more , which can occur in about 1% of patients. Consequently, frequent monitoring of white blood cells (done weekly for the first 6 months and every 2 weeks thereafter, then once a month after a year) is required in the US, and clozapine is generally reserved for patients who have responded inadequately to other drugs. Newer SGAs (see table Second-Generation Antipsychotics Second-Generation Antipsychotics*) provide some of the benefits of clozapine without the risk of agranulocytosis and are generally preferable to conventional antipsychotics for treatment of an acute episode and for prevention of recurrence. However, in a large, long-term, controlled clinical trial, symptom relief using any of 4 SGAs (olanzapine, risperidone, quetiapine, ziprasidone) was no greater than that with perphenazine, a conventional antipsychotic with anticholinergic effects. In a follow-up study, patients who left the study prematurely were randomized to one of the 3 other study SGAs or to clozapine; this study demonstrated a clear advantage of clozapine over the other SGAs. Hence, clozapine seems to be the only effective treatment for patients who have failed treatment with a conventional antipsychotic or an SGA. However, clozapine remains underused, probably because of lower tolerability and need for continuous blood monitoring. Lumateperone is the newest SGA for treatment of schizophrenia in adults. It appears to improve psychosocial function with fewer metabolic and motor side effects. It should not be used in older patients with dementia-related psychosis, in whom it carries an increased risk of death. Other side effects include sedation and dry mouth. Weight gain, hyperlipidemia, and elevated risk of type 2 diabetes are the major adverse effects of SGAs. Thus, before treatment with SGAs is begun, all patients should be screened for risk factors, including personal or family history of diabetes, weight, waist circumference, blood pressure, and fasting plasma glucose and lipid profile. Those found to have or be at significant risk of metabolic syndrome Metabolic Syndrome Metabolic syndrome is characterized by a large waist circumference (due to excess abdominal fat), hypertension, abnormal fasting plasma glucose or insulin resistance, and dyslipidemia. Causes... read more may be better treated with ziprasidone or aripiprazole than the other SGAs. Patient and family education regarding symptoms and signs of diabetes Symptoms and Signs Diabetes mellitus is impaired insulin secretion and variable degrees of peripheral insulin resistance leading to hyperglycemia. Early symptoms are related to hyperglycemia and include polydipsia... read more , including polyuria, polydipsia, weight loss, and diabetic ketoacidosis (nausea, vomiting, dehydration, rapid respiration, clouding of sensorium), should be provided. In addition, nutritional and physical activity counseling should be provided to all patients when they start taking an SGA. All patients taking an SGA require periodic monitoring of weight, body mass index, and fasting plasma glucose and referral for specialty evaluation if they develop hyperlipidemia or type 2 diabetes. Antidepressants/selective serotonin-norepinephrine reuptake inhibitors Neuroleptics, also known as antipsychotic medications, are used to treat and manage symptoms of many psychiatric disorders. They fall into two classes: first-generation or "typical" antipsychotics and second-generation or "atypical" antipsychotics. Both first and second-generation antipsychotics are used in various neuropsychiatric conditions. These include attention-deficit hyperactivity disorder (ADHD), behavioral disturbances in dementia, geriatric agitation, depression, eating disorders, personality disorders, insomnia, generalized anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder (PTSD), and substance use and dependence disorders. For many of these conditions, the evidence for their use is equivocal. This activity reviews the indications and contraindications of neuroleptics and highlights the role of the interprofessional team in the safer prescription of these drugs. Objectives: Identify the mechanism of action of neuroleptics.Describe the adverse effects of neuroleptics.Summarize the indicated and off-label use of neuroleptics.Explain interprofessional team strategies for enhancing care coordination and communication to advance the safer prescribing of neuroleptics and improve outcomes. Access free multiple choice questions on this topic. Neuroleptics, also known as antipsychotic medications, are used to treat and manage symptoms of many psychiatric disorders. They fall into two classes: first-generation or "typical" antipsychotics and second-generation or "atypical" antipsychotics." Neuroleptic drugs block dopamine receptors in the nervous system.First-generation antipsychotics were developed initially in the 1950s for the treatment of psychosis (e.g., schizophrenia).[1] In addition to psychotic illnesses, they have also been FDA-approved for treating and managing acute mania, agitation, bipolar disorder, Tourette syndrome, and hyperactivity. Due to the poorly tolerated and often irreversible adverse effects of first-generation antipsychotics, the second class of neuroleptics (second-generation antipsychotics), were established in the 1980s. These second-generation antipsychotics have been FDA-approved to treat and manage psychosis as well as treatment-resistant schizophrenia, bipolar disorder, schizoaffective disorder, agitation, and irritability/agitation. By 2001, 96% of the neuroleptics prescribed to new users were second-generation.Beyond their FDA-approved uses, both first and second-generation antipsychotics also are used in several neuropsychiatric conditions that are currently considered off-label. These include attention-deficit hyperactivity disorder (ADHD), behavioral disturbances in dementia, geriatric agitation, depression, eating disorders, personality disorders, insomnia, generalized anxiety disorder, obsessive-compulsive disorder, post-traumatic stress disorder (PTSD), and substance use and dependence disorders. For many of these conditions, the evidence for their use is inconclusive. With increasing off-label use and high costs, it is important to evaluate the evidence for their emergent use in these medical and psychiatric conditions.[2][3][4][5]In first-generation antipsychotic medications, the postsynaptic blockade of dopamine D2 receptors in the mesolimbic system of the central nervous system (CNS) is the mechanism of action. Evidence suggests strong antagonism of D2 receptors in both striatal and cortical areas, a higher association between D2 receptor binding and its potency, and a consistent requirement of 65% D2-receptor occupancy for antipsychotic efficacy in functional imaging studies. The nonspecific localization of dopamine binding throughout the central nervous system (CNS) is consistent with the risk of movement disorders (Parkinson's disease) and prolactinemia.[6][7][8]Second-generation antipsychotics differ from first-generation by transiently occupying D2 receptors, followed by rapidly dissociating, allowing normal dopamine neurotransmission. They also have fast D2 dissociation, antagonistic properties on the 5HT2A receptor, and 5HT1A agonism. Second-generation antipsychotics have fewer side effects and are generally considered safe in adult and older populations. These differences account for the normal prolactin levels, lessened cognitive deficits, and preclusion of extrapyramidal symptoms.[9]Most first-generation antipsychotic medications are available in oral formulations. Several are also available in injectable intramuscular formulations, which are useful in the treatment of psychotic agitation. Clinicians sometimes use intravenous formulations of haloperidol and droperidol to treat psychosis, agitation, or delirium in acute medical settings. Long-acting, decanoate preparations of haloperidol and fluphenazine are deliverable via intramuscular injection one to two times per month, which is useful for nonadherent patients with daily oral dosing.Second-generation antipsychotics are available in oral form. Additionally, aripiprazole is available as an intramuscular injection (immediate release) for use in acute settings, and olanzapine, risperidone, paliperidone, and aripiprazole are available in the form of long-acting injectables for use in nonadherent patients. The injectable form is for use in older and non-compliant patients, so the steady dose of the antipsychotic is available without any withdrawal effects.[10]First-generation or typical antipsychotic drugs are either high potency or low potency, based on the amount of drug necessary to minimize patient symptoms. Specific dosing of individual agents will not be covered in this class overview article.In addition to their activity at D2 antagonists, first-generation antipsychotics exert significant effects on 5-HT2A, alpha-1, histaminic, and muscarinic receptors, which correspond to their side-effect profiles. These pharmacological differences are the basis for the classification of first-generation antipsychotics as either high or low-potency medications.[11][12][13]The high-potency, first-generation antipsychotics, such as fluphenazine, trifluoperazine, haloperidol, loxapine, pimozide, perphenazine, and thiothixene, are dosed in the range of one to tens of milligrams. They display low activity at histaminic and muscarinic receptors. They are associated with weight gain, sedative effects, or anticholinergic activity. They have a high risk of extrapyramidal side effects (dystonia, bradykinesia, rigidity, tremor, neuroleptic malignant syndrome, and tardive dyskinesia due to dopamine receptor hypersensitivity and hyperprolactinemia.Low-potency, first-generation antipsychotics like chlorpromazine and thioridazine are dosed in hundreds of milligrams and have high histaminic and muscarinic activity with a corresponding heightened prevalence of dizziness, sedation, and anticholinergic effects (dry mouth, urinary retention, constipation), but carry a lower risk of extrapyramidal side effects.Due to the fewer extrapyramidal and anticholinergic effects, second-generation antipsychotics are the first-line treatment for psychotic disorders (e.g., schizophrenia). Second-generation antipsychotics have correlations with weight gain, type 2 diabetes mellitus, metabolic syndrome, fatigue/drowsiness, sedation, and QTc prolongation. Among the second-generation antipsychotics, clozapine is most effective in reducing psychotic symptoms and suicidality. The use of clozapine is mostly for treatment-resistant schizophrenia. However, due to the significant adverse side effect of agranulocytosis, clozapine is reserved for severe cases of psychotic disorders after a patient has had an insufficient response to two adequate trials of other antipsychotic medications.[14]Because of their wide-ranging adverse effects, neuroleptic medications may cause or aggravate some conditions. They are contraindicated in patients with liver damage, coronary artery disease, cerebrovascular disease, parkinsonism, bone marrow depression (i.e., clozapine), severe hypotension or hypertension, coma, or severely depressed states. They should be used cautiously in people with seizure disorders, diabetes mellitus, glaucoma, prostatic hypertrophy, peptic ulcer disease, and chronic respiratory diseases.[15]First-generation (typical) antipsychotic drugs qualify for therapeutic drug monitoring primarily for compliance control and to avoid extrapyramidal reactions by keeping chronic exposure to minimal adequate blood levels. For clozapine, drug safety with regards to agranulocytosis is another reason to use therapeutic drug monitoring.With second-generation antipsychotics (risperidone, olanzapine, quetiapine, ziprasidone, and aripiprazole), the rationale to monitor drug levels is debatable. Positron emission tomography (PET) enables measurement of the occupancy of dopamine D2 receptors and reveals receptor occupancy interacts better with plasma concentrations than with doses of the antipsychotics. There is a consistent requirement of 65% D2 receptor occupancy for antipsychotic efficacy in functional imaging studies. Regarding plasma levels related to therapeutic effects, there are established ideal concentrations for clozapine (350 ng/mL to 600 ng/mL), risperidone (20 ng/mL to 60 ng/mL), and olanzapine (20 ng/mL to 80 ng/mL) but not for the other second-generation antipsychotics. In conclusion, the evidence is growing that drug monitoring may improve efficacy and safety in patients treated with the new antipsychotic drugs, especially when patients do not respond or develop side effects under therapeutic doses.An isolated overdose of neuroleptics is rarely fatal. Toxicity results from blockade of some or all of the following receptors: dopamine (extrapyramidal symptoms), alpha-1 (orthostatic hypotension, reflex tachycardia), muscarinic (anticholinergic symptoms), and histaminic (sedation).[16]The extrapyramidal symptoms include acute dystonia (tongue protrusion, facial grimacing, trismus, and oculogyric crisis) and akathisia (feeling of inner restlessness). The anticholinergic effects include tachycardia, dry mucous membranes, dry skin, decreased bowel sounds, and delirium. These symptoms can be managed with diphenhydramine 25 mg to 50 mg IV/IM or benztropine 1 mg to 2 mg IV/IM. ECG changes such as sinus tachycardia and QT prolongation can result from neuroleptic toxicity. With QTc prolongation of more than 500 ms, treatment with magnesium 2 gm to 4 gm IV over 10 minutes is indicated.The most life-threatening emergency associated with the use of neuroleptics is neuroleptic malignant syndrome.[17][18] This syndrome can occur from a single dose, increasing dose, or the same dose. It is mostly associated with first-generation antipsychotics. Still, it can also occur to a lesser degree with the second-generation antipsychotics, antiemetics (metoclopramide, promethazine), and the withdrawal of anti-Parkinson medication. Symptoms typically develop over 1 to 3 days, mortality rates are 5% to 20%, and the majority of deaths occur due to complications of muscle rigidity. Clinical characteristics of neuroleptic malignant syndrome include the tetrad of altered mental status, muscular rigidity, hyperthermia, and autonomic instability. Management involves stopping the causative agent, supportive care with fluid resuscitation and cooling measures and directed medical therapy of dantrolene (skeletal muscle relaxant) at 0.25 mg/kg to 2 mg/kg IV every 6 to 12 hours with a max dose of 10 mg/kg/day or bromocriptine (dopamine agonist) at 2.5 mg by mouth every 6 to 8 hours with a max dose 40 mg/day.Neuroleptic drugs are beneficial for the management of behavioral disorders. The newer generation is safer, but they still can cause adverse effects that include weight gain, hyperlipidemia, and metabolic syndrome. All interprofessional healthcare team members should encourage the patient to eat a healthy diet, exercise regularly, and avoid smoking. Frequent measurement of body weight, ECG, and lipids is necessary. Because of the disease and side effects, compliance with medication is not high.[19] And the patient should be educated about the importance of compliance and the adverse effects that one may encounter. Prescribing clinicians need to carefully examine each patient's case to decide if they will benefit from therapy with neuroleptic medications. Nurses should provide counseling on dosing and regarding potential adverse events, which can also be reinforced by the pharmacist. Pharmacists should also examine the patient's medication record to determine the potential for any drug-drug interactions and report any possible issues to the prescriber. This interprofessional approach will lead to increased therapeutic benefit with a lower potential for adverse events [Level 5]While prescribing high-risk drugs, the patient should always closely follow up with the psychiatrist and continuous involvement of internists to look for any signs of serious adverse effects (agranulocytosis, metabolic syndrome, extrapyramidal syndrome, QT interval prolongation).Review Questions1.Cussotto S, Clarke G, Dinan TG, Cryan JF. Psychotropics and the Microbiome: a Chamber of Secrets…. Psychopharmacology (Berl). 2019 May;236(5):1411-1432. [PMC free article: PMC6598948] [PubMed: 30806744]2.Das B, Rawat VS, Ramasubbu SK, Kumar B. Frequency, characteristics and nature of risk factors associated with use of QT interval prolonging medications and related drug-drug interactions in a cohort of psychiatry patients. Therapie. 2019 Dec;74(6):599-609. [PubMed: 31053339]3.Pandey S, Dash D. 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