declined from 76% to 31% as a share of all uses from 1994 to 2007, while substantial increases in use occurred for bipolar affective disorder (5% to 39%) and depression (8% to 15%). The fraction of atypical antipsychotic use for indications with insufficient evidence of efficacy increased from 32% in 1994 to 38% in 2007, representing 26 billion prescriptions and $7.3 billion dollars in expenditures in 2007. During 2007, primary care physicians accounted for 21% of visits where an antipsychotic was used, as compared with psychiatrists (77%) or physicians from other specialties (2%). Antipsychotic use in settings of insufficient evidence was similar among primary care physicians, internists, and psychiatrists. CONCLUSIONS: The scope and costs of this expansion, due to both clinical innovation and overuse, demonstrate the importance of efforts to limit the clinical application of antipsychotics to settings of sufficient evidence.

PMH49
ADULT ADHD IN THE UNITED STATES: A COMPARISON OF 2 METHODS TO ESTIMATE PREVALENCE
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OBJECTIVES: To validate adult ADHD prevalence estimates in a US health care claims database. METHODS: A commercial research database (MarketScan® from Thomson Reuters; employer clients only) was used to estimate the annual prevalence of adult ADHD from 2002 to 2007. Patients (18-64 years) diagnosed with ADHD (ICD-9) on at least 2 occasions within 12 months were counted in each year they had a diagnostic/direct claim indicating ADHD. These prevalence rates were compared with rates from a US epidemiological study which estimated adult ADHD prevalence using clinical interviews of respondents (18-44 years) from the 2005 National Comorbid- dity Survey replication (NCS-R; Kessler et al., 2006). RESULTS: In MarketScan® the prevalence of diagnosed and treated ADHD in US adults was 1.24 per 1000 members in 2002, increasing annually to more than triple in 2007 (4.02 per 1000). The proportion of females: males with ADHD increased yearly. Prevalence per 1000 grew faster among 18-24 than 25-64 year olds. ADHD with hyperactivity was more prevalent than ADHD without hyperactivity. In contrast, the NCS-R study reported a higher ADHD prevalence (44 per 1000) in 2003 in the 18-44 group, with more males than females diagnosed with ADHD. Prevalence estimates from our study increased when correcting for differences between studies in age range (18-64 versus 18-44 years; 4.59 per 1000) and number of diagnoses (2 vs 1; 5.03 per 1000). In the NCS-R study, only 10.8% of individuals who were diagnosed received treatment. Thus, the number of treated patients in the NCR-S study (4.796 per 1000) is close to the 4.02 per 1000 reported from the claims database. CONCLUSIONS: The estimated prevalence of adult ADHD in diagnosed and treated patients based on claims data is similar to that based on clinical interviews, validating the use of claims data to estimate prevalence.

PMH10
RISK OF INJURY IN ADULTS WITH ATTENTION-DEFICIT HYPERACTIVITY DISORDER
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OBJECTIVES: To evaluate the risk of injury associated with attention-deficit hyperactivity disorder (ADHD) using information from an employee database. METHODS: Using the MarketScan® Commercial Claims and Encounters Database from Thomson Reuters, patients aged 18 to 64 years and diagnosed with ADHD (N = 31,752) were matched by similarity in demographics and data availability 3:1 to controls without ADHD (N = 95,256) or 1:1 to controls diagnosed with depression (N = 29,965). Patients with ADHD were also stratified into compliant (Medication Possession Ratio [MPR] ≥ 0.8, N = 8,654), partially compliant (0.3 ≤ MPR < 0.8, N = 5,213) and non-compliant (MPR < 0.3, N = 4,007) cohorts. Risk of injury was compared between groups for January to December 2006. Multivariate analyses controlled for treatment differences between groups that remained after matching. RESULTS: Injury rates were higher in the ADHD group than in the non-ADHD control group (21.6% vs 15.7%, p < 0.001) and depression group (21.4% vs 20.5%, p = 0.0008), and higher in the compliant group than in the partially compliant (22.8% vs 20.3, p = 0.0004) and noncompliant (22.8% vs 17.8%, p < 0.001) groups. In multivariate analyses, risk of injury was higher in the ADHD group than in the non-ADHD (1.3194 odds ratios [OR], p < 0.001) and depression control groups (1.1263 OR, p < 0.01) and higher in compliant/partially compliant patients than in noncompliant patients (1.2633 and 1.664 OR, respectively, p < 0.01). Comorbid depression, anxiety and substance abuse predicted a higher risk of injury in the ADHD versus control groups (p < 0.01) and in compliant/partially compliant patients versus noncompliant patients (p < 0.01), with magnitude equal to or exceeding that of the ADHD group alone. CONCLUSIONS: Patients with ADHD had higher risk of injury than similarly matched patients without ADHD or with depression, suggesting important implications for workplace safety and liability.

PMH11
RISK ASSESSMENT OF POISONING WITH DRUGS OF THERAPEUTIC IMPORTANCE BY TOXICOLOGICAL IMPACT
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OBJECTIVES: Toxicological studies were carried out with the aim to determine the health hazards, treatment and outcome of therapeutic drug overdose. MEIHDS: All therapeutic drug poisoned patients admitted to a tertiary care South Indian hospital were evaluated for predisposing factors manner of exposure, severity at admission, treatment, hospitalization period, clinical status at discharge, in order to implement medication safety programs. RESULTS: A total of 200 patients were admitted with therapeutic drug overdose. A total of 153 cases intentionally ingested the drugs, while 29 had overdose during routine therapy, and 18 cases were accidental. Depression was the major predisposing factor for intentional self harm. Most of the poisoning with therapeutic drugs occurred due to intentional self harm n = 133 (76.3%). Other manner of exposures include overdose during therapeutic use n = 14 (8.5%) or accidental exposure n = 18 (9%). There was a significant (p < 0.001) association of employment and occupational status on the manner of exposure. The mean GCS, APACHE II scores, PMR and PSS for all the patients was 8.46 ± 3.6; 20.63 ± 5.0; 38.58 ± 16.1 and 2.98 ± 0.79 (Mean ± SD) respectively. The mean GCS score was significantly (P = 0.033) different between various class of poisoning. The APACHE II scores were not significantly (P > 0.05) different between various drug classes. The mean severity scores was significantly (P = 0.016) different between various class of drugs. The treatment provided was empirical and symptomatic with differing decontamination procedures. The average hospitalization period was 6.68 ± 4.9 days. Mortality was lowest with benzodiazepines (7.1%), followed by opioids (11.8%), antihypertensives (12.7%) and antidepressants (13.3%). MORTALITY: 15 patients died during hospital stay. CONCLUSIONS: The extent of harm caused due to therapeutic drug poisoning can be minimized by systematically estimating the severity at triage and providing treatment as per standard guidelines.

PMH12
A META-ANALYSIS OF EFFICACY AND SAFETY OF PREGABALIN AND CLONAZEPAM IN THE TREATMENT OF ANXIETY DISORDERS
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OBJECTIVES: The purpose of this study was to evaluate the relative efficacy and safety of pregabalin and clonazepam in the treatment of anxiety disorders. METHODS: A meta-analysis was performed with randomized clinical trials (RCTs) where pregabalin or clonazepam were used for any anxiety disorders. Effectiveness was assessed with the Hamilton Anxiety Rating Scale (HAM-A); safety with the frequency and type of adverse events (AEs). RESULTS: Six RCTs were included in both pregabalin and clonazepam groups. Odds ratios and weighted means differences (WMD) were calculated. Both, fixed and random effects models were employed in the analysis. RESULTS: From 1989 abstracts, we obtained 40 RCT; 23 were excluded (unsuitable designs, insufficient outcome data, no placebo control) leaving 17. Seven pregabalin studies were used to evaluate its effectiveness and safety; four clonazepam studies for effectiveness and six for safety. Clonazepam studies included panic disorder and social phobia. Six of seven pregabalin studies were in generalized anxiety disorder and one for social anxiety disorder. We found that both drugs significantly diminished anxiety levels after four to 32 weeks of treatment. Among, clonazepam studies the frequency of AEs was higher than pregabalin by nearly 50%; with pregabalin, less than 30%. We didn’t find any head-to-head studies with pregabalin and clonazepam and no statistical difference in anxiety level reduction was distinguished between drugs, WMD in HAM-A: 1.29 (95% CI = 1.13, 1.54). However, clonazepam showed higher AE rates than pregabalin in somnolence (OR 0.34; 95% CI 0.44-0.66, headache (OR 0.50; 0.34-0.74), blurred vision (OR 0.36, 0.13-0.98) and cognitive impairment (OR 0.25; 0.09-0.72). CONCLUSIONS: Clonazepam and pregabalin are effective in diminishing anxiety levels in several anxiety disorders, although clonazepam seems to cause a higher frequency of AEs.