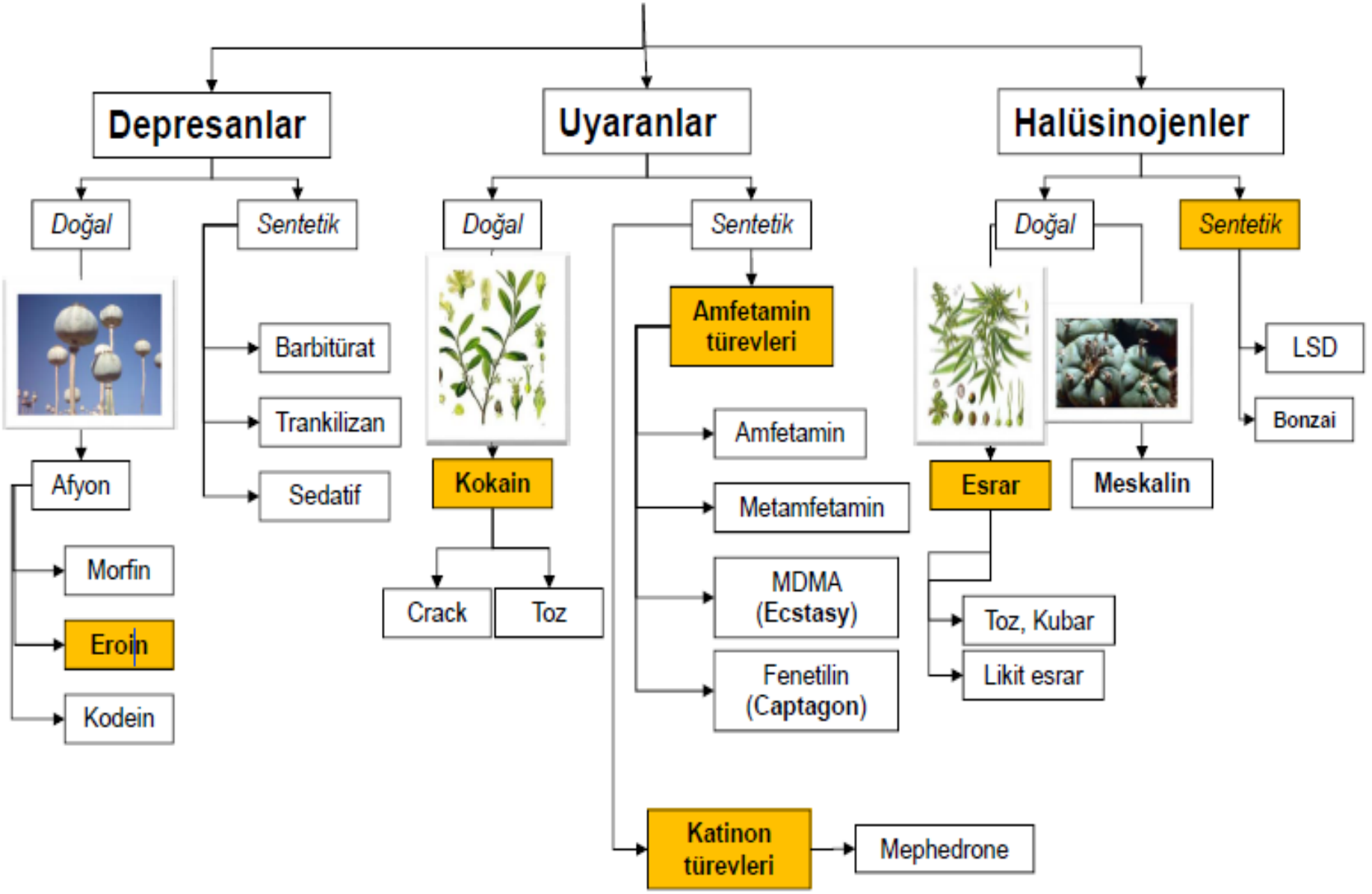
SANTRAL SİNİR SİSTEMİ UYARICILARININ ÖLÜMCÜL YAN ETKİLERİ (SAK)

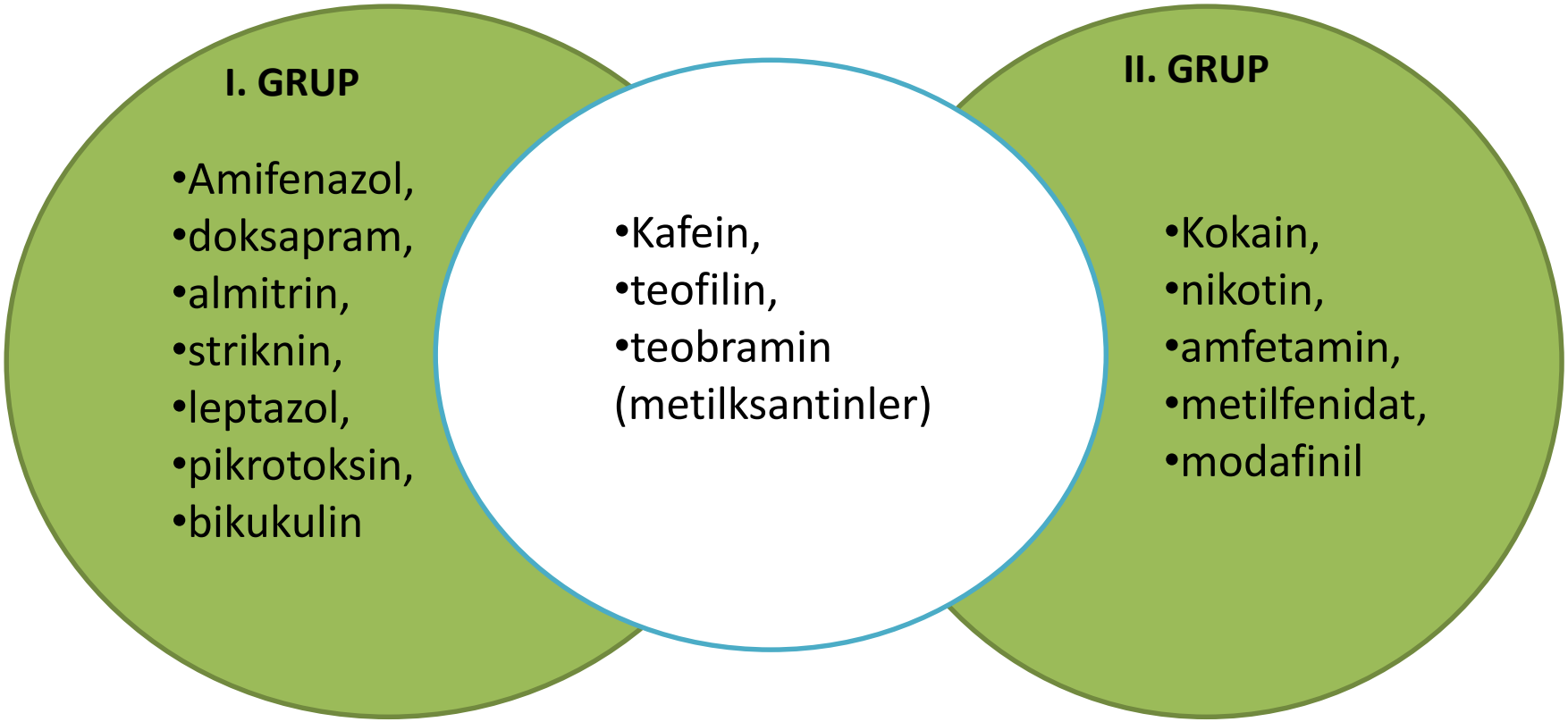
Dr. Kasım TURGUT
Adıyaman Üniversitesi Tıp Fakültesi

SOKAK İLACLARI



❑ SSS üzerinden vücuttaki birçok mekanizmayı etkileyen ilaçlar. 2 gruba ayrılır;

- ➢ Konvülzan ve solunum uyarıcılar (I)
- ➢ Psikomotor stimülanlar (II)



Temel mekanizmalar

- NA, dopamin, serotonin gibi katekolaminleri serbestleştirir ve re-uptake inhibisyonu (amfetamin ve türevleri, kokain)
- Fosfodiesteraz inhibisyonu (metilksantinler)
- GABA, Glisin antagonizması (striknin, bikukulin, piktotoksin)
- Gangliyonik stimülasyon(nikotin)

Kullanım şekilleri

- SSS uyarıcıları bir çok farklı hastalığın tedavisinde kullanılır.
  - DEHB
  - Norkolepsi,
  - Obezite (iştah gezici),
  - Lokal anestetik
  - Depresyon?
  - Strok rehabilitasyonu?
  - Travmatik beyin yaralanmalarında?

1880'li yıllarda kokainin bugünkü yan etkileri tam olarak bilinmiyor ve kontrolsüzce her alanda kullanılıyordu. Bu masum reklam broşüründe kokain içeren bir diş ağrısı ilacının tanıtımı yapılıyor.

- Yasadışı kullanım sebepleri;
  - Uyanıklık ve konsantrasyon sağlayıcı
  - Doping
  - Keyif alıcı
  - Bağımlılık

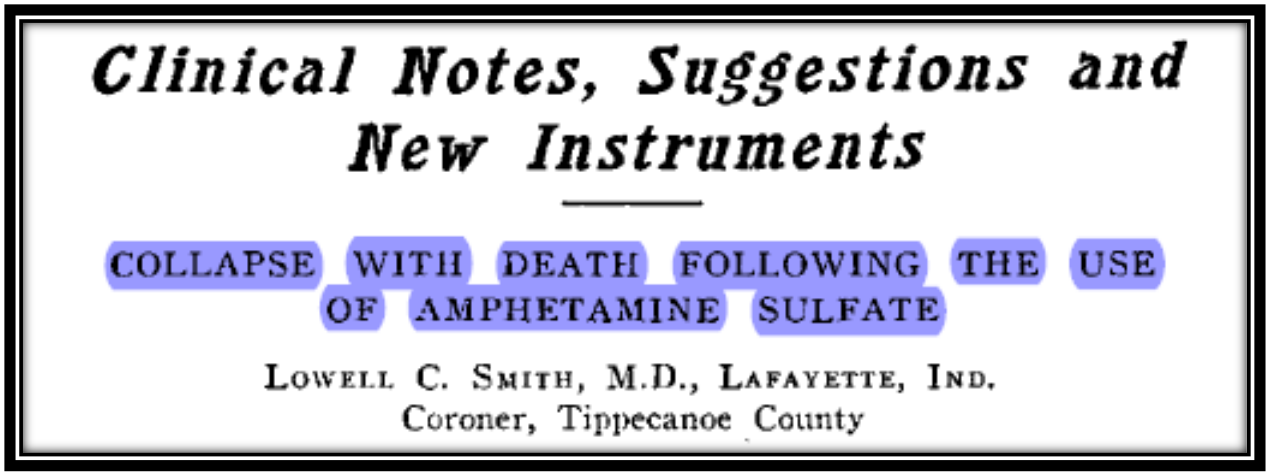
- ABD de 1.5 milyon kokain kullanıcısı
- 353.000 metamfetamin kullanıcısı
- Opiyat ve esrardan sonra 3. sıklıkta
- Aynı şekilde yasal veya yasadışı olarak suistimali en fazla yapılan ilaçlardan olup toplam oran;
  - Avrupada-% 2.7
  - Amerikada-% 4.5
- % 43 reçetesiz kullanım

Yan etkiler

- Büyüme ve gelişme geriliği
  - Özellikle DEHB tanılı çocuklarda boy ve kilo geriliği
- Uyku düzensizliği
- İştahsizlık
- Tikler
- Psikoz
- Epilepsi atak

Ciddi yan etkiler

- Kardiyovasküler ve nörolojik sistem üzerinde ölümcül yan etkileri vardır. En fazla bilinen;
  - Koroner vazospazm ve MI
  - Hipertansiyon,
  - Taşikardi,
  - Ani kardiyak ölüm,
  - Nörolojik serebral vazospazm
  - İskemik ve hemorajik SVO



Clinical Notes, Suggestions and New Instruments

COLLAPSE WITH DEATH FOLLOWING THE USE OF AMPHETAMINE SULFATE

LOWELL C. SMITH, M.D., LAFAYETTE, IND.
Coroner, Tippecanoe County

Amfetamin kullanımına bağlı ilk ölüm 1939 yılında sınav başarısını artırmak amaçlı tekrarlayan alım olan

- Ayrıca intihar amaçlı aşırı doz alımına bağlı ölümler azımsanmayacak kadar çoktur.

- SSS uyarıcıları kaynaklı ölümler için yapılan çalışmada dünya genelinde;
  - amfetamin kaynaklı ölüm oranı %0.58,
  - kokain kaynaklı ölüm oranı ise %0.32

Main causes of death and standardized mortality ratios among 428 individuals age 20–59 who inject amphetamine. Main causes of death according to ICD-10.

Causes of death	n	%	SMR	95% CI	ICD-10
Certain infectious and parasitic diseases	28	7	38.3	23.8-52.7	A00-B99
Neoplasms	48	11	3.2	2.3-4.1	C00-D48
Endocrine, nutritional and metabolic diseases	7	2	4.7	0.9-8.4	E00-E90
Diseases of the nervous system	8	2	5.5	1.7-9.3	G00-G99
Diseases of the circulatory system	67	16	5.4	4.1-6.8	I00-I99
Diseases of the respiratory system	17	4	12.7	6.7-18.7	J00-J99
Diseases of the digestive system	24	6	10.0	6.0-14.0	K00-K93
Diseases of the skin and subcutaneous tissue	1	0.2	–	–	L00-L99
Mental and behavioral disorders	52	12	31.2	22.7-39.7	F00-F99
Unknown causes of death	14	3	9.4	4.5-14.3	R00-R99
External causes of morbidity and mortality	162	38	12.7	10.8-14.7	V01-Y99
- Intentional self-harm (suicide)			5.3	3.3-7.3	X60-X84
- Men			5.0	3.0-7.1	
- Women			7.0	0.1-13.9	
- Poisoning, accidental or of undetermined intent			32.0	25.4-38.6	
- Transport accidents			8.0	6.8-14.5	V01-V99
Total	428[a]	100.0	8.3	7.5-9.1	

Table 2 Direct cause of death.

Cause of death (%)	*Male (n = 285)*	*Female (n = 86)*	*All (n = 371)*
Methamphetamine toxicity	17	16	17
Combined drug toxicity	51	53	51
Cardiovascular	15	11	14
Cerebrovascular	4	13*	6
Injury	9	8	9
Pulmonary	5	4	5
Hanging	5	4	5
Other	7	6	7

Table 5 Major organ pathology.

Type of pathology (%)	*Males (n = 172)*	*Females (n = 48)*	*All (n = 220)*
Cardiovascular pathology	55	48	54
Atherosclerosis	43	26*	39
Mild	20	15	19
Moderate	3	2	3
Severe	17	7	15
Unspecified	1	2	1
Sites of atherosclerosis	40	17*	35
Coronary arteries	14	17	14
Aorta			
Cardiomegaly	18	6	16
Hypertrophy	13	11	12
Ischaemic heart disease	6	2	5
Cerebrovascular pathology†	19	26	20
Pulmonary pathology‡	42	42	42
Hepatic pathology§	61	60	61
Renal pathology¶	12	13	13

KARDİYAK YAN ETKİLER

- Kan basıncını yükseltme
- Nabız hızını arttırma
- Vazospazm

} katekolamin artışı

- Proinflamatuvar ürünler aracılığıyla vaskülit
- QT aralığını uzatma ve torsade pointes
- Platelet agregasyonu ve trombüs oluşumu

- Akut miyokard enfarktüsü
- Disritmi
- Hipertansiyon-diseksiyon, kapak yetmezlikleri
- Kardiyomiyopati
- Myokardit
- Subendokardiyal iskemi ve fibrozis

White SR. Amphetamine toxicity. Semin Respir Crit Care Med. 2002;23(1):27-36.

Westover and Halm *BMC Cardiovascular Disorders* 2012, **12**:41
http://www.biomedcentral.com/1471-2261/12/41

BMC Cardiovascular Disorders

RESEARCH ARTICLE Open Access

Do prescription stimulants increase the risk of adverse cardiovascular events?: A systematic review

Arthur N Westover[1,2*] and Ethan A Halm[2,3]

Abstract

Background: There is increasing concern that prescription stimulants may be associated with adverse cardiovascular events such as stroke, myocardial infarction, and sudden death. Public health concerns are amplified by increasing use of prescription stimulants among adults.

Methods: The objective of this study was to conduct a systematic review of the evidence of an association bet

Results: Ten population-based observational studies which evaluated prescription stimulant use with cardiovascular outcomes were reviewed. Six out of seven studies in children and adolescents did not show an association between stimulant use and adverse cardiovascular outcomes. In contrast, two out of three studies in adults found an association.

Conclusions: Findings of an association between prescription stimulant use and adverse cardiovascular outcomes are mixed. Studies of children and adolescents suggest that statistical power is limited in available study populations, and the absolute risk of an event is low. More suggestive of a safety signal, studies of adults found an increased risk for transient ischemic attack and sudden death/ventricular arrhythmia. Interpretation was limited due to differences in population, cardiovascular outcome selection/ascertainment, and methodology. Accounting for confounding and selection biases in these studies is of particular concern. Future studies should address this and other methodological issues.

to differences in population, cardiovascular outcome selection/ascertainment, and methodology. Accounting for confounding and selection biases in these studies is of particular concern. Future studies should address this and other methodological issues.

- Watts DJ, McCollester L: Methamphetamine-induced myocardial infarction with elevated troponin I. Am J Emerg Med 2006, 24:132–134.
- Ibrahim M, Hasan R, Awan M. Cocaine-induced coronary stent thrombosis. Exp Clin Cardiol. 2013 Winter;18(1):e57-9.
- Oo, Z. T., & Kyaw, H. (2020). Role of Stent Versus Thrombolysis in Management of Cocaine-Induced ST-Elevation Myocardial Infarction. *Cureus*, *12*(8), e9654
- Thompson J, Thompson JR: Acute Myocardial Infarction Related to Methylphenidate for Adult Attention Deficit Disorder. J Emerg Med 2007,38(1):18–21.
- Gandhi PJ, Ezeala GU, Luyen TT, Tu TC, Tran MT: Myocardial infarction inan adolescent taking Adderall. Am J Health Syst Pharm 2005, 62:1494–1497.
- George AK, Kunwar AR, Awasthi A: Acute myocardial infarction in a young male on methylphenidate, bupropion, and erythromycin. J Child Adolesc Psychopharmacol 2005, 15:693–695.
- Chen JP: Methamphetamine-associated acute myocardial infarction and cardiogenic shock with normal coronary arteries: refractory global coronary microvascular spasm. J Invasive Cardiol 2007, 19:E89–E92.

Heart, Lung and Circulation (2016) 25, 325–332
1443-9506/04/$36.00
http://dx.doi.org/10.1016/j.hlc.2015.10.019

REVIEW

The Cardiac Complications of Methamphetamines

Elizabeth D. Paratz, MBBS [a*], Neil J. Cunningham, MBBS, FACEM [b], Andrew I. MacIsaac, MD [a]

Taylor & Francis

Expert Opinion on Drug Safety

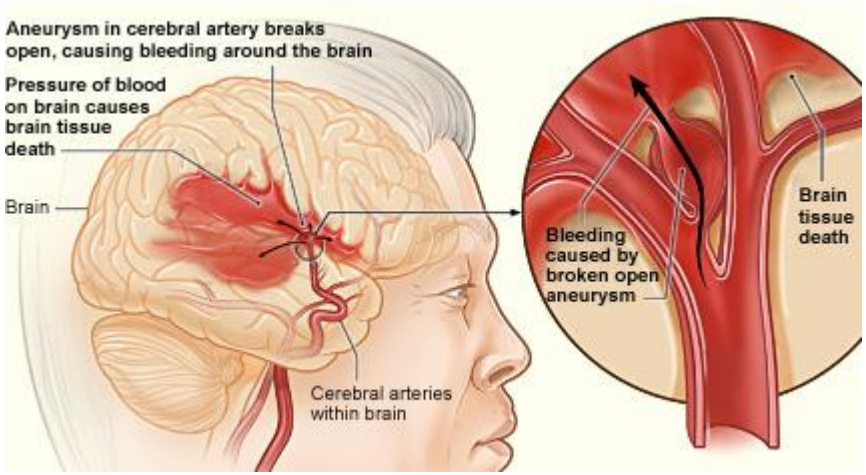
ISSN: 1474-0338 (Print) 1744-764X (Online) Journal homepage: http://www.tandfonline.com/loi/ieds20

An update on the safety of psychostimulants for the treatment of attention-deficit/hyperactivity disorder

Annabeth P. Groenman, Lizanne J.S. Schweren, Andrea Dietrich & Pieter J. Hoekstra

NÖROLOJİK YAN ETKİLER

- Çoğunlukla kardiyovasküler sistem üzerinde oluşturdukları değişikliklerle
- Burada en önemli iki etken;
  - uyarıcılara bağlı gelişen tansiyon ve nabız değişimleri
  - kişinin öncesinde anevrizma, AVM gibi vasküler bir lezyonunun olmasıdır



- Ayrıca uzun süreli stimulan kullanımı beyin damarlarında

İltihaplanma ➕ ateroskleroz ➡ anevrizma

- Otopsi çalışmalarında beyinde vaskülit ve aşırı ateroskleroz görülmüş ve bu tekrarlayan tansiyon değişimlerine bağlanmıştır.

Beadell NC, et al. The deleterious effects of methamphetamine use on initial presentation and clinical outcomes in aneurysmal subarachnoid hemorrhage. J Neurosurg. 2012;117(4):781-6

- Uyarıcılara bağlı çoğunlukla **hemorajik svo** görülür;
  - Çoğunlukla AVM veya anevrizma kaynaklı
  - genellikle parankim içine
  - SAK tek başına veya parankim içine kanamaya eşlik edebilir
- **İskemiler**;
  - beyin sapı veya serebral hemisferlerde
  - Temel mekanizma vaskülit zemininde oluşan trombüsler
- **Serebral ödem**
- **Epileptik atak**

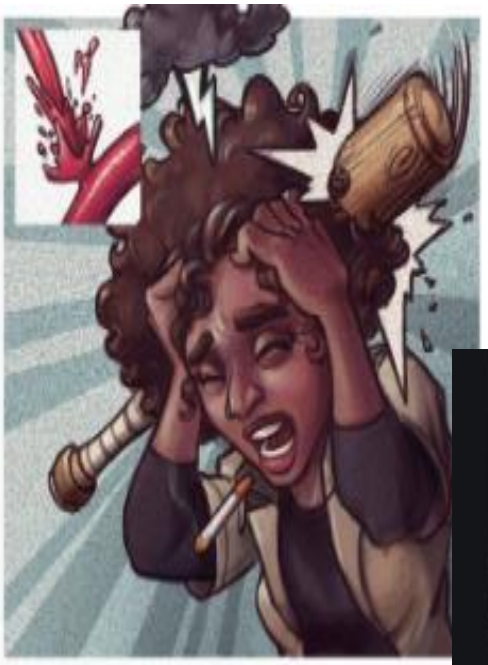
SUICIDE BY INGESTION OF AMPHETAMINE SULFATE

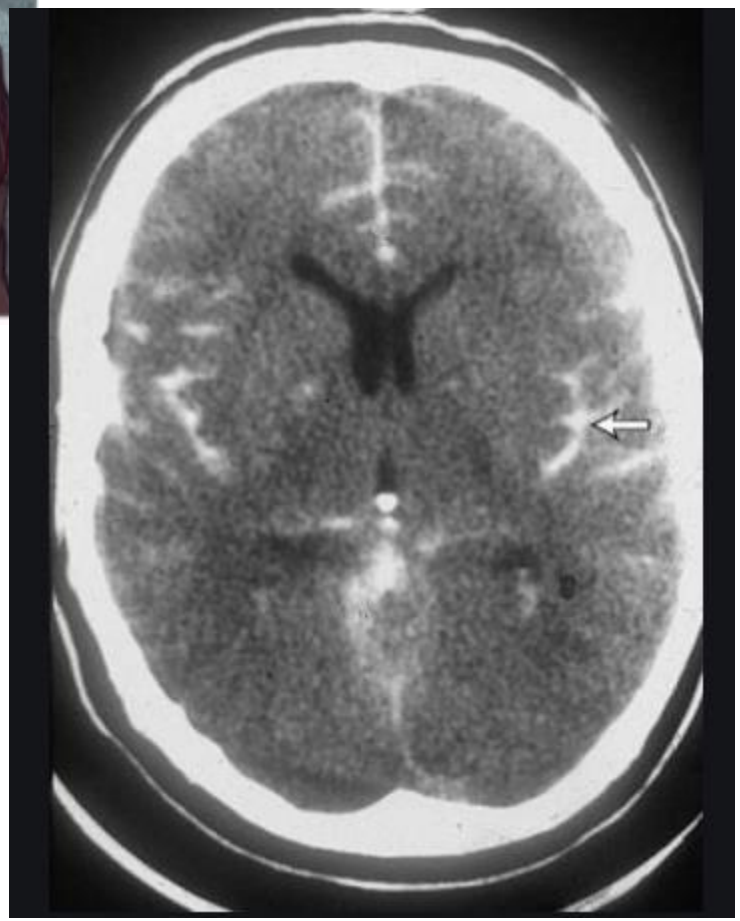
MAJOR O. L. GERICKE
MEDICAL CORPS, ARMY OF THE UNITED STATES

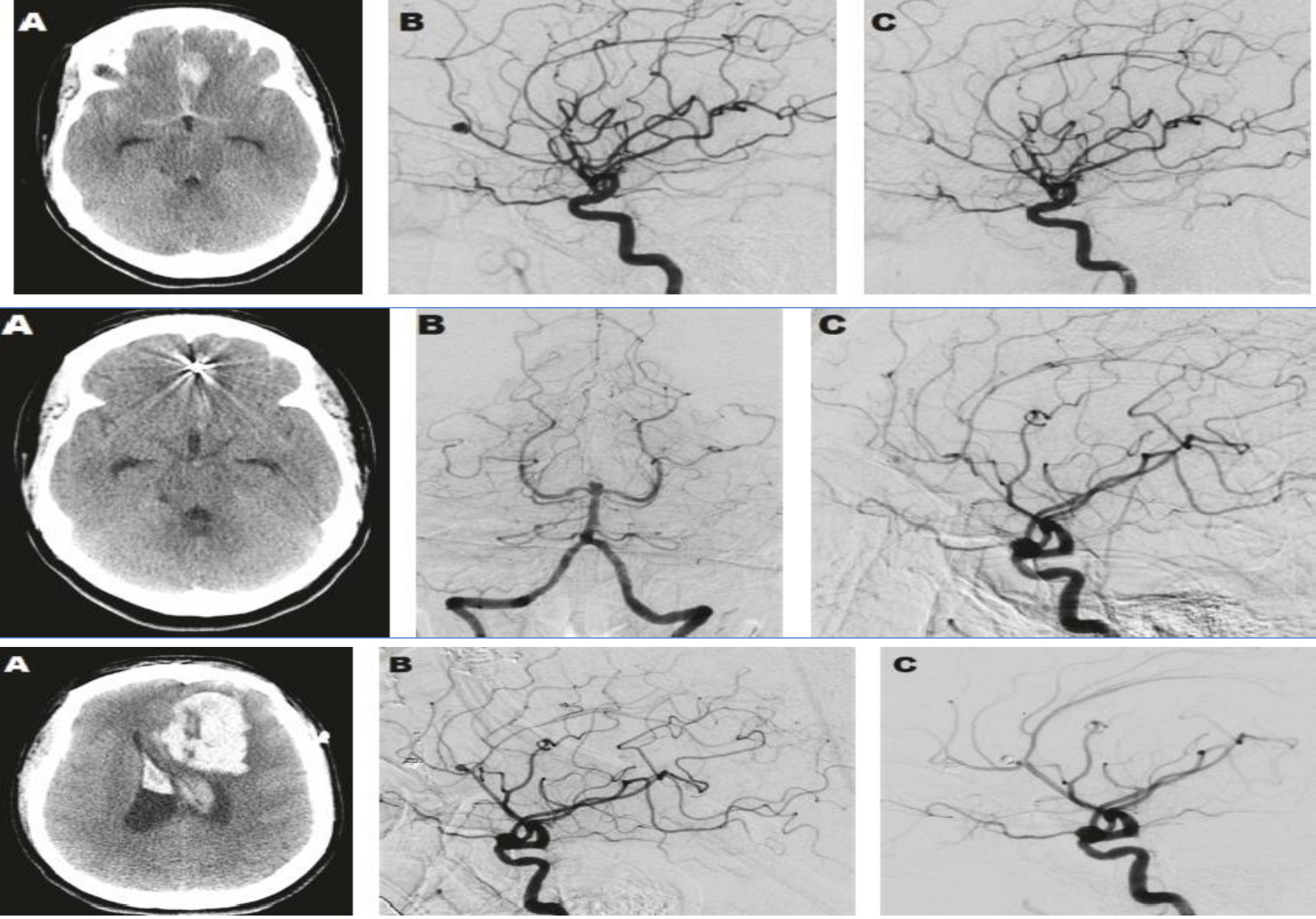
SUMMARY

Death of a 36 year old man followed the ingestion of an estimated 120 mg. of amphetamine sulfate taken with suicidal intent. The immediate cause of death was subdural and subarachnoid hemorrhage of the parietal and occipital lobes.

- *Gericke OL (1945) Suicide by ingestion of amphetamine sulphate.JAMA 128:1098–1099*
- *Brust JC, Richter RW (1977) Stroke associated with cocaine abuse? N Y State J Med 77:1473–1475*







Fowler J, et al. Impact of Methamphetamine Abuse: A Rare Case of Rapid Cerebral Aneurysm Growth with Review of Literature. Case Rep Neurol Med. 2018;2018:1879329

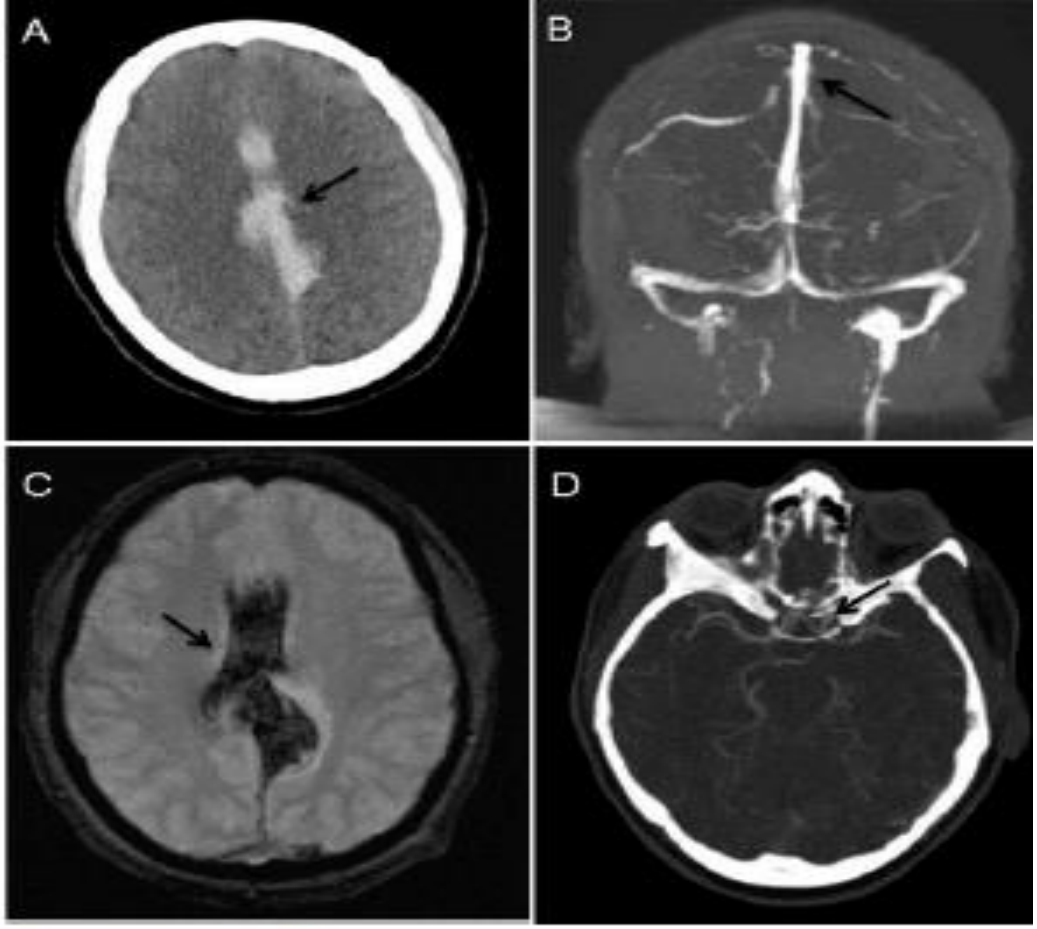


Figure 1 A) Non-contrast head CT revealing extensive interhemispheric and subarachnoid haemorrhage (arrow (B) MRV of the Head demonstrating patency of the sagittal sinus in the region of the haemorrhage (arrow) and absence of dural sinus thromboses.
(C) MRI brain, hemoflash sequence, corroborating the area of haemorrhage (arrow). (D) CT angiogram of head demonstrating a small, incidental left supraclinoid internal carotid artery aneurysm (arrow). No obvious vasculitic changes or abnormalities in the areas of intracranial haemorrhage seen.

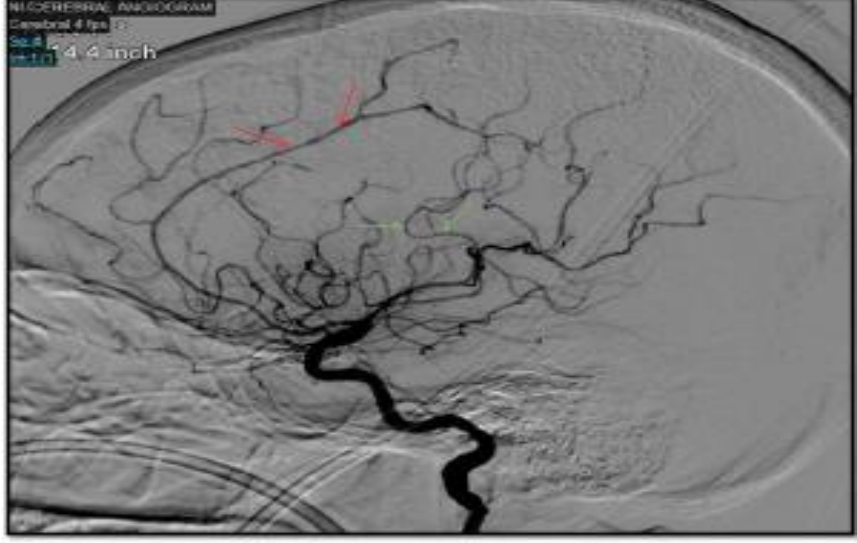


Figure 2 Sagittal view of cerebral angiogram showing areas of luminal irregularity and beading in left anterior cerebral artery (red arrows) and left middle cerebral artery (green arrows).

Nagele EP, et al. Interhemispheric subdural and subarachnoid haemorrhage in a patient with amphetamine-induced vasculitis. BMJ Case Rep. 2017:bcr2017222918.

Amphetamine abuse and intracranial haemorrhage

Neil Buxton FRCS(Ed) Norman S McConachie FRCR[1]

J R Soc Med 2000;**93**:472–477

SUMMARY

Amphetamines taken by any route can cause cerebral vasculitis and intracranial haemorrhage. 8 cases were seen in a neurosurgical unit over 3.5 years. The published work indicates that those who experience these complications, mainly young adults, have poor outcomes.

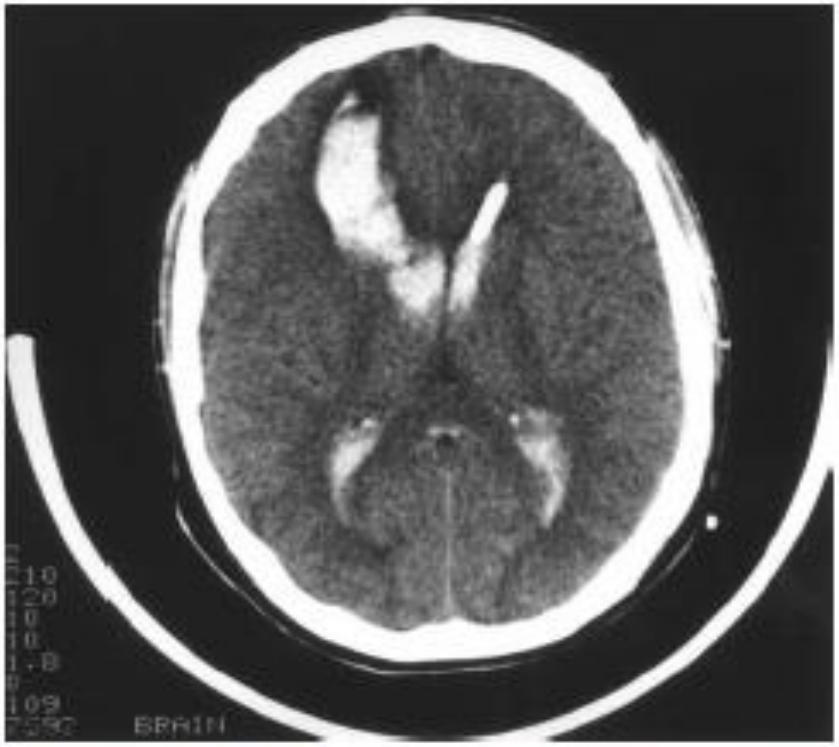


Figure 1 **Large frontal haemorrhage extending into the lateral ventricle**

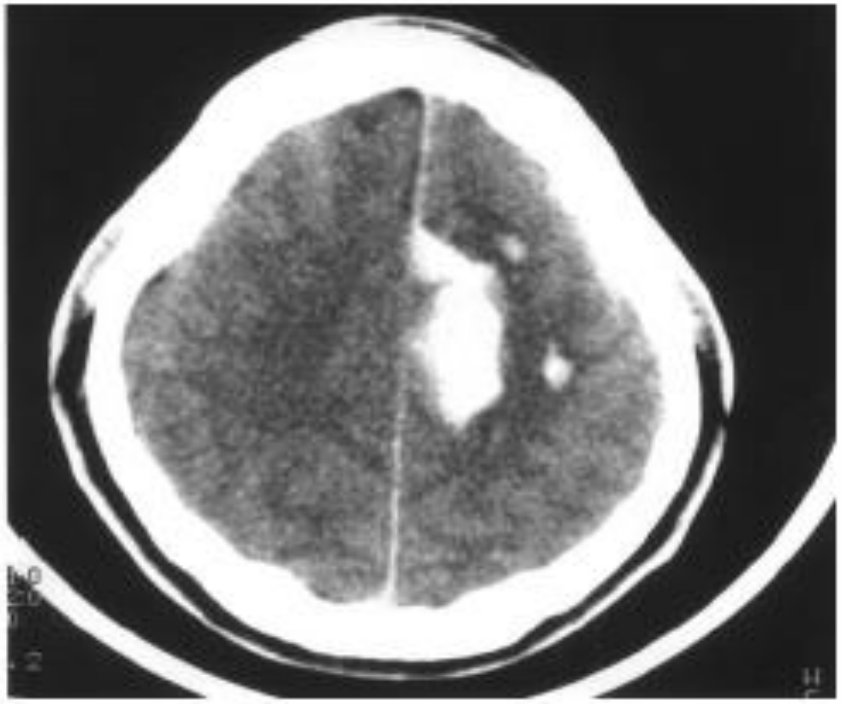


Figure 2 **Left cerebral haematoma involving cingulate gyrus and frontoparietal white matter**

ELSEVIER

Forensic Science International 129 (2002) 104–109

Forensic Science International

www.elsevier.com/locate/forsciint

A case of fatal hemorrhage in the cerebral ventricles following intravenous use of methamphetamine

Fumio Moriya*, Yoshiaki Hashimoto

Neurol Med Chir (Tokyo) 31, 49~52, 1991

Subarachnoid and Intracerebral Hemorrhage Associated with Necrotizing Angiitis Due to Methamphetamine Abuse

—An Autopsy Case—

Subarachnoid hemorrhage in a patient taking phentermine for weight loss

Jonathan A. Bain, Jeremy S. Dority, and Aaron M. Cook


"PEOPLE WHO TOOK THIS PICKLE BASED PILL FOR THREE MONTHS DIDN'T GET CANCER!"
"...DURING THOSE THREE MONTHS"
DOT

Contents lists available at ScienceDirect

Drug and Alcohol Dependence

journal homepage: www.elsevier.com/locate/drugalcdep

Review

Cocaine use and risk of stroke: A systematic review☆

L. Sordo[a,b,c,*] B.J. Indave[d] G. Barrio[e] L. Degenhardt[f,g,h] L. de la Fuente[a,b] M.J. Bravo[a,b]

Background: Both cocaine use and strokes impact public health. Cocaine is a putative cause of strokes, but no systematic review of the scientific evidence has been published.

Methods: All relevant bibliographic-databases were searched until January 2014 for articles on the epidemiological association between cocaine use and strokes. Search strings were supervised by expert librarians. Three researchers independently reviewed studies for inclusion and data extraction following STROBE recommendations. Quality appraisal included study validity and bias. Both ischemic and hemorrhagic strokes were considered.

Results: Of 996 articles, 9 were selected: 7 case–control studies (CCS) and 2 cross-sectional (CSS) studies. One CCS (aOR = 6.1; 95% CI: 3.3–11.8) and one CSS (aOR = 2.33; 95% CI: 1.74–3.11) showed an association between cocaine and hemorrhagic strokes. The latter study also found a positive relationship with ischemic stroke (aOR = 2.03; 95% CI: 1.48–2.79). Another CCS found the exposure to be associated with stroke without distinguishing between types (aOR = 13.9; 95% CI: 2.8–69.4). One forensic CCS found that deaths with cocaine-positive toxicology presented a 14.3-fold (95% CI: 5.6–37) and 4.6-fold (95% CI: 2.5–8.5) increased risk of atherosclerosis compared to opioid-related deaths and hanging-deaths respectively. One CCS did not provide an aOR but found a statistically significant association between cocaine and hemorrhagic stroke. Three CCS and one CSS did not find any relationship between cocaine and strokes. Inadequate control for confounding was not uncommon.

Conclusions: Epidemiological evidence suggests that cocaine use increases the risk of stroke. Larger, more rigorous observational studies, including cohort approaches, are needed to better quantify this risk, and should consider stroke type, hypertension variation, frequency/length of cocaine use, amphetamines co-use, and other factors.

Major Risk Factors for Aneurysmal Subarachnoid Hemorrhage in the Young Are Modifiable

Joseph P. Broderick, MD; Catherine M. Viscoli, PhD; Thomas Brott, MD; Walter N. Kernan, MD; Lawrence M. Brass, MD; Edward Feldmann, MD; Lewis B. Morgenstern, MD; Janet Lee Wilterdink, MD; Ralph I. Horwitz, MD; for the Hemorrhagic Stroke Project Investigators

Background and Purpose—To identify risk factors for subarachnoid hemorrhage (SAH) and intracerebral hemorrhage, we designed a case-control study of men and women 18 to 49 years of age (the Hemorrhagic Stroke Project [HSP]). This report focuses on SAH.

Methods—Patients were recruited from 44 hospitals in the United States. Cases with SAH must have had a ruptured aneurysm documented by angiography or surgery. Two controls, identified by random digit dialing and matched to each patient for age, sex, race, and telephone exchange, were sought for each case subject.

Results—Between 1994 and 1999, 425 patients with SAH were enrolled in HSP, and 312 cases met the criteria for aneurysmal SAH. The present analyses also included 618 matched controls. Of the 312 cases, 66% were current cigarette smokers compared with 30% of controls (adjusted odds ratio [OR], 3.73; 95% CI, 2.67 to 5.21). Cocaine use within the previous 3-day period was reported by 3% of cases and no controls (bivariate exact OR, 24.97; 95% exact CI, 3.95 to ∞; adjusted estimate not calculable). Other independent risk factors in the multivariable model included hypertension (adjusted OR, 2.21; 95% CI, 1.48 to 3.29), low body mass index (OR, 1.59; 95% CI, 1.08 to 2.35), primary family history of hemorrhagic stroke (OR, 3.83; 95% CI, 1.73 to 8.46), caffeine in pharmaceutical products (OR, 2.48; 95% CI, 1.19 to 5.20), lower educational achievement (OR, 2.36; 95% CI, 1.44 to 3.87), and nicotine in pharmaceutical products (adjusted estimate not calculable).

Conclusions—Aneurysmal SAH may be largely a preventable disease among the young and middle-aged because several prevalent risk factors can be modified by medication (eg, hypertension) or behavioral change (eg, cigarette smoking, cocaine use). The association of caffeine and nicotine in pharmaceutical products and aneurysmal SAH warrants further study. (*Stroke*. 2003;34:1375-1381.)

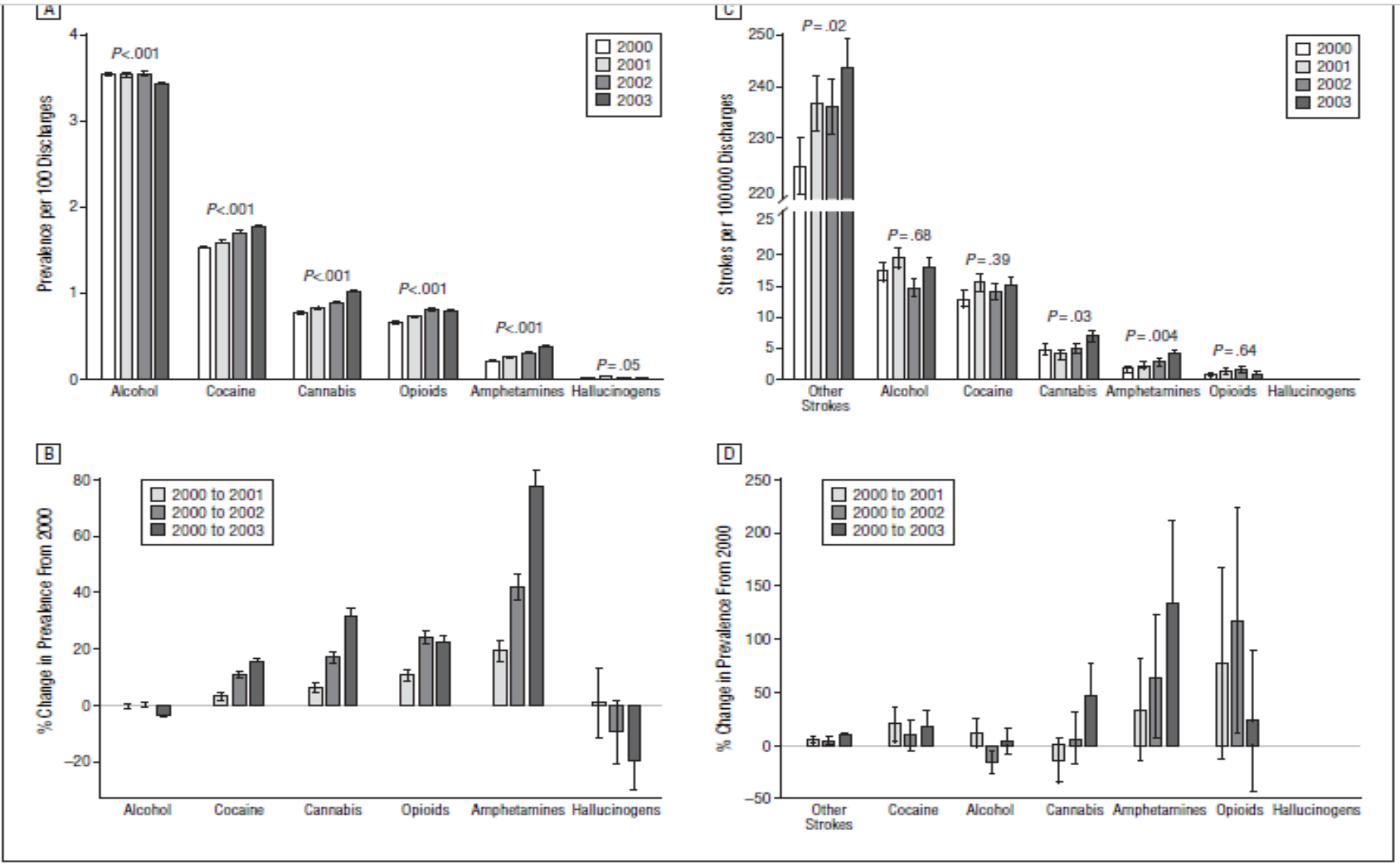


Figure 1. Trends in drug abuse and stroke associated with drug abuse in discharges of persons aged 18 to 44 years from Texas hospitals, from January 1, 2000, to December 31, 2003: annual prevalence rate of abuse of various drugs (A), percentage change in prevalence rates from 2000 (B), incidence rates of stroke associated with history of abusing various drugs (C), and percentage change in incidence rates from 2000 (D). All data are given plus or minus the standard error. The *P* values were obtained using the Cochrane-Armitage test for trend (2-tailed). In C and D, "other strokes" indicate all strokes not associated with alcohol, cocaine, cannabis, amphetamines, opioids, or hallucinogens.

Intracerebral hemorrhage in cocaine users

Sheryl Martin-Schild, **Karen C. Albright**, **Hen Hallevi**, **Andrew D. Barreto**, **Maria Philip**, **Vivek Misra**, **James C. Grotta**, and **Sean I. Savitz**

Abstract

Background—Cocaine is a cause of intracerebral hemorrhage (ICH), but there are no large studies that have characterized the location, pathology, and outcome of patients with cocaine-associated ICH.

Methods—We performed a retrospective analysis of all patients admitted to our stroke service from 2004 to 2007 who had non-traumatic ICH and urine drug screens positive for cocaine and compared them with similar patients who had a negative drug screen for cocaine.

Results—We identified 45 patients with cocaine-associated ICH and 105 patients with cocaine-negative ICH. There were no significant differences in age or gender but there was a significantly higher incidence of African-American patients in the cocaine positive group. Cocaine-associated ICH patients had higher admission blood pressures, significantly more subcortical hemorrhages, and higher rates of intraventricular hemorrhage (IVH) compared to patients with cocaine-negative ICH. Cocaine-positive patients had worse functional outcome, defined as an mRS>3 at the time of discharge (OR 4.90, 95% CI 2.19–10.97), and were less likely to be discharged home or to inpatient rehab. Patients with cocaine-associated ICH were nearly 3 times more likely to die during their acute hospitalization when compared to cocaine-negative patients.

Conclusion—Recent cocaine ingestion is associated with hemorrhages that occur more frequently in subcortical locations, have a higher risk of IVH, and carry a poor prognosis compared to patients with cocaine-negative, spontaneous ICH.

Impact of Acute Cocaine Use on Aneurysmal Subarachnoid Hemorrhage

Tiffany R. Chang, MD; Robert G. Kowalski, MBBCh, MS; Filissa Caserta, MSN, ACNP-BC, CNRN; Juan Ricardo Carhuapoma, MD; Rafael J. Tamargo, MD; Neeraj S. Naval, MD

Background and Purpose—Acute cocaine use has been temporally associated with aneurysmal subarachnoid hemorrhage (aSAH). This study analyzes the impact of cocaine use on patient presentation, complications, and outcomes.

Methods—Data of patients admitted with aSAH between 1991 and 2009 were reviewed to determine impact of acute cocaine use (C). These patients were compared with aSAH patients without recent cocaine exposure (NC) in relation to their presentation, complications such as aneurysmal rerupture and delayed cerebral ischemia, and outcomes including hospital mortality and functional outcome.

Results—Data of 1134 aSAH patients were reviewed; 142 patients (12.5%) had associated cocaine use. Cocaine users were more likely to be younger (mean age: C, 49±11; NC, 53±14; $P<0.001$). There were no differences in rates of poor-grade Hunt and Hess (4–5); (C, 21%; NC, 26%; $P>0.05$), associated intraventricular hemorrhage (C, 56%; NC, 51%; $P>0.05$), or hydrocephalus on admission Head CT (C, 49%; NC, 52%; $P>0.05$). Aneurysm rerupture incidence was higher among cocaine users (C, 7.7%; NC, 2.7%; $P<0.05$). The association of cocaine use with higher risk of delayed cerebral ischemia (C, 22%; NC, 16%; $P<0.05$) was not significant after correcting for other factors. Cocaine users were less likely to survive hospitalization compared with nonusers (mortality: C, 26%; NC, 17%; $P<0.05$); the adjusted odds of hospital mortality were 2.9 times higher among cocaine users ($P<0.001$). There were no differences in functional outcomes between the 2 groups.

Conclusions—Acute cocaine use was associated with a higher risk of aneurysm rerupture and hospital mortality after aSAH. (*Stroke*. 2013;44:1825-1829.)

ORIGINAL RESEARCH

Methamphetamine use is an independent predictor of poor outcome after aneurysmal subarachnoid hemorrhage

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ABSTRACT

Background Clinical outcomes of methamphetamine users with aneurysmal subarachnoid hemorrhage (aSAH) are unknown.

Objective To analyze differences in presentation, in-hospital morbidity, and outcomes between methamphetamine users and non-users.

Methods All 472 patients included in the Barrow Ruptured Aneurysm Trial from 2003 to 2007 were reviewed. Patients with 1- and 3-year follow-up were included in this analysis (n=398). Methamphetamine users were identified as patients who provided a history of methamphetamine use on admission or tested positive on urine toxicology testing. Methamphetamine users were compared with non-users using univariate analysis. Outcomes were then analyzed using multivariate logistic regression models for demographic characteristics, medical comorbidities, radiographic and clinical presentation, and vasospasm.

Results Thirty-one patients (7.8%) were identified as methamphetamine users in this cohort. Methamphetamine users were younger than non-users (mean age 42.8 vs 55 years, p<0.001). In multivariate logistic regression models, methamphetamine use was an independent predictor of poor Glasgow Outcome Scale score at both 1 year (OR=5.02; 95% CI 1.03 to 24.48; p<0.05) and 3 years (OR=7.18; 95% CI 1.73 to 29.87; p=0.007). Other independent predictors in this model included older age, clinical vasospasm, diabetes, and aneurysm size. Cocaine and tobacco use were not significantly associated with poor outcome in our cohort. Methamphetamine use was not significantly associated with vasospasm, higher Fisher or Hunt and Hess grade, or intraparenchymal hemorrhage/intraventricular hemorrhage.

Conclusions Methamphetamine users have significantly worse outcomes at 1 and 3 years following aSAH. Further analysis is necessary to understand the pathological response associated with methamphetamine use in this setting.

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