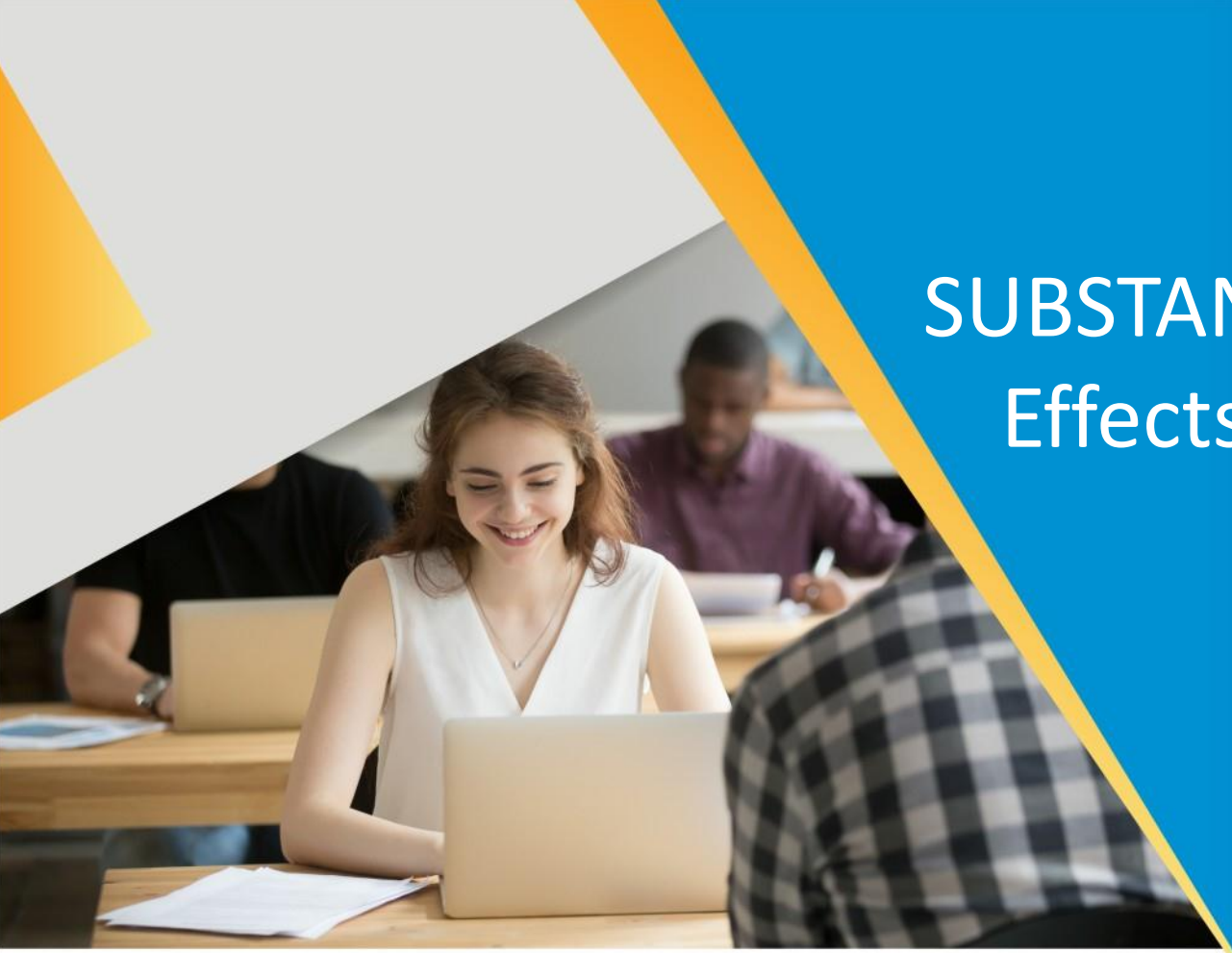

SUBSTANCE ABUSE : Effects on the gut.

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Commonly abused drugs/ substances

- Alcohol (21.4%) was the primary substance used (apart from tobacco) followed by cannabis (3.0%) and opioids (0.7%) – 2004 NHDS.
- 76% of the opioid users currently injected buprenorphine, 76% injected heroin, 70% chasing (inhalation of brown sugar) and 64% using propoxyphene. Most drug users concomitantly used alcohol (80%).

- Brown sugar – heroin (impure form) – morphine diacetate
- Ganja (hashish)– cannabis/ marijuana/ cannabinoid/ grass/ reefer
- Charas - hashish

- Alcohol.
- Opioids.
- Cocaine and crack.
- Cannabis.
- Amphetamines and stimulants.
- Sedative hypnotics.
- Hallucinogens.
- Inhalants/ nicotine and others.

- According to the World Drug Report, of 81,802 treatment seekers in India in 2004-2005, 61.3% reported use of opioids, 15.5% cannabis, 4.1% sedatives, 1.5% cocaine, 0.2% amphetamines and 0.9% solvents.

GUT effects

- The trauma to the GUT caused by introducing large amounts of drugs is something that can last well beyond active drug use.
- The negative effect of drug abuse on the digestive system is mainly due to the deterioration of the mucous membrane – ulcers, bleeding, inflammation.
- Effect on nutritional intake.
- Effect on motility.

Mechanical effects

- Most common – dehydration ; affecting secretion of enzymes, including saliva.
- The process usually begins with nausea, irrespective of whether the subject is abusing amphetamines or laxatives.
- Vomitting, diarrhoea or constipation are common effects.
- GI bleed is an uncommon side effect of overdose or abuse.
- GERD – common, related to the drugs, medications or abuse

Nutritional effects

- Mal-nutrition
- Micronutrient deficiency.
- Fast food.
- Obesity.
- Lack of exercise.

Alcohol

- Even moderate alcohol intake (20-40 g/day) can cause fatty liver or alcoholic hepatitis, both of which are reversible.
- Cirrhosis occurs in 10-20% of chronic alcohol dependent patients (consuming >120-180 g alcohol/day) and is not reversible.
- Nutritional and ethnic factors contribute.

How much?

- Pequignot (1958) and Pequignot et al. (1974) estimated that the average cirrhogenic alcohol consumption is 180 g of ethanol per day consumed regularly for approximately 25 years; the risk is increased five times at a level of consumption between 80 and 160 g/day and 25 times if daily ethanol consumption exceeds 160 g.
- Regularly = most days of the week for most weeks of the year.

What constitutes heavy drinking?

- Daily intake of alcohol as low as 40 g by men and 20 g by women resulted in an increased incidence of cirrhosis in a well-nourished population.
- Turner proposed that to qualify as moderate, one's alcohol consumption should not exceed 0.8 g/kg of body weight (bw) per day (an absolute limit of 80 g of alcohol) or an average of 0.7 g/kg bw in a 3-day period.
- ? Total lifetime intake of 100kg….?
- AASLD – agrees that anything more than 30g/day is dangerous.

AH

- Acute alcoholic hepatitis – worse prognosis short term, usually associated with a “binge”.
- Severity may be judged by MDF, Lille model, Glasgow score, MELD, etc.
- AST>ALT, usually AST:ALT ratio is more than 2, and ALT is rarely more than 5xULN.
- MDF > 32 – high risk of dying (40-50%); GAHS more than 8 – poor outcome.

Cirrhosis

Diagnosis

- Cirrhosis – how to suspect?
  - Jaundice.
  - Portal hypertension.
  - Low platelet count.
  - Ascites.
- Imaging.
- Blood tests – low albumin, high INR.

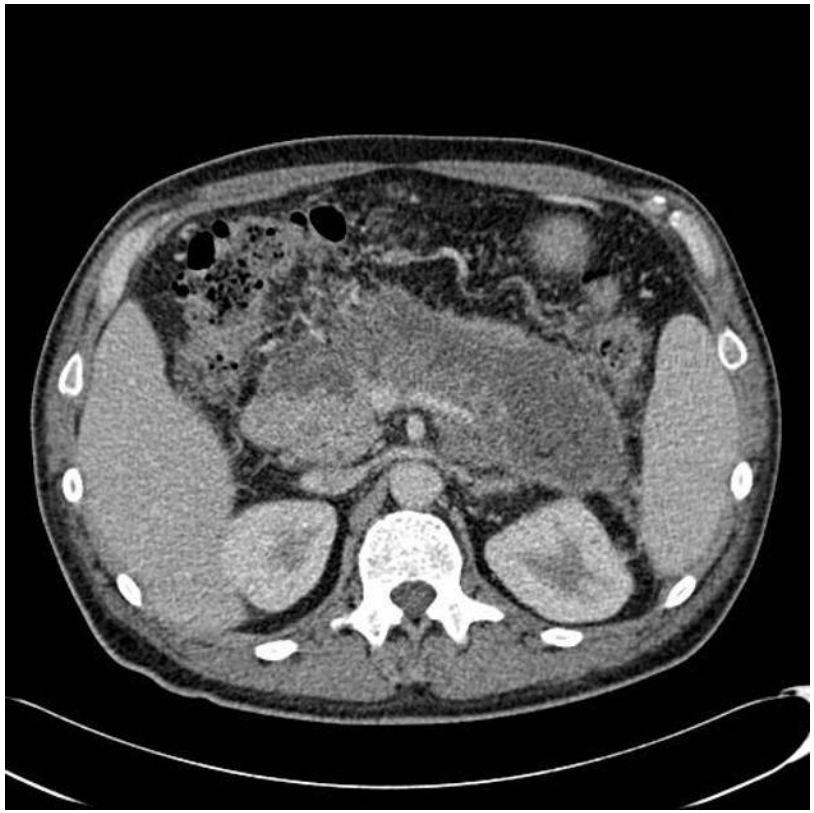
Alcoholic pancreatitis

- Overuse of alcohol is a major cause of acute and chronic pancreatitis in both developed and developing countries.
- Prolonged overconsumption of alcohol for 5–10 years typically precedes the initial attack of acute alcoholic pancreatitis.
- Genetic?
- Ethnic?
- Malnutrition?
- Specific susceptibility/ trigger?

Acute Pancreatitis

- Acute attack.
- Systemic effects.
- Organ failure.
- High mortality.

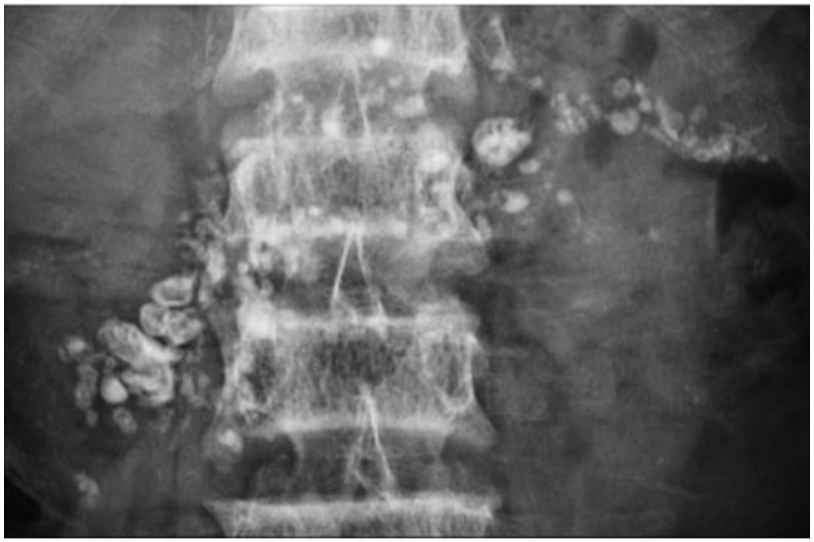
Acute pancreatitis



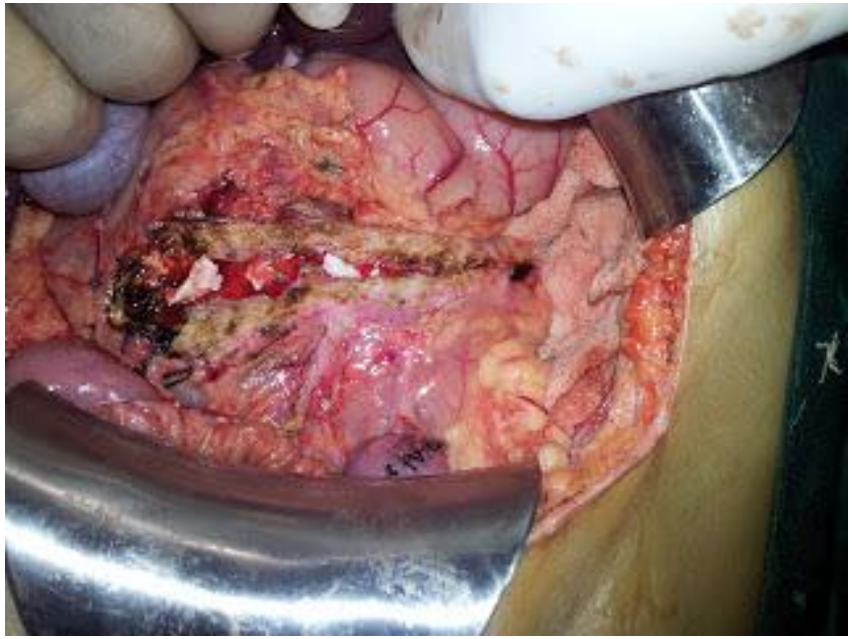
Chronic pancreatitis

- As a result of repeated injury and scarring.
- Anatomical changes.
- Pain.
- High morbidity.
- ? Surgery/ drainage.
- Abdominal pain coupled with malabsorption/ maldigestion and diabetes resulting from the exocrine and endocrine insufficiency.

Chronic calcific pancreatitis



Pancreatic Calcification



Suspect Pancreatitis if...

- Acute abdominal pain.
- Unexplained shock.
- Abdominal distension.
- Persistent vomiting.
- Get USG abdomen if suspicion is high.

Alcohol abuse and GUT

- Leaky gut.
- Reduced efficacy of prostaglandins.
- ? Decrease in enzyme output.
- “Leaky gut syndrome” - diarrhea, excessive flatulence, unexplained fatigue, unexplained fever, heartburn, ongoing or recurrent abdominal pain, recurrent bladder infections, hemorrhoids, food allergies, frequent hunger.
- Affects nutrition.
- Reduced gut immunity.

Effects of leaky gut

- Malnutrition.
- Intestinal infections.
- Weight loss inspite of adequate nutrition.
- Anemia.
- Occult GI bleed.

Carcinogenic…?

Definite increased risk….

- Liver cancer.
- Stomach cancer.
- Esophageal cancer.
- Pancreatic cancer.

Possible increase in susceptibility…

- Colon cancer.
- Gall bladder cancer.

OPIODS

Narcotic bowel syndrome!

- Characterized by the progressive and somewhat paradoxical increase in abdominal pain despite continued or escalating dosages of narcotics.
- There may be previous h/o narcotic use, but can happen without.
- NBS should be distinguished from opioid-induced bowel disorder, which results from the effects of opioids on gastrointestinal motility and secretion.

- Opiates are very well known for causing constipation, even at their normal dosage.
- Long-term abuse of painkillers means that many users will need to rely on laxatives to move the bowels, and increased risk of piles and fissures.
- nausea, bloating, vomiting, abdominal distention can happen due to opiods.
- Opiods lead to an increase in the risk of UGI cancers.

- Opioids cause spasm of the sphincter of Oddi.
- Long term users have a higher incidence of gall stones.
- Higher incidence of pancreatitis.

Cocaine

- Peptic ulcer – with high incidence of perforation.
- Mesenteric ischemia and gangrene.
- GI bleed.
- Ischemic colitis (long term use) – leading to strictures.

- Mechanism:
  - Vasoconstriction.
  - Ischemic ulceration.
  - Platelet aggregation.
  - Altered motility, increased intragastric pressure (associated with the smoking of crack, which may in part be due to increased air swallowing and breath holding).

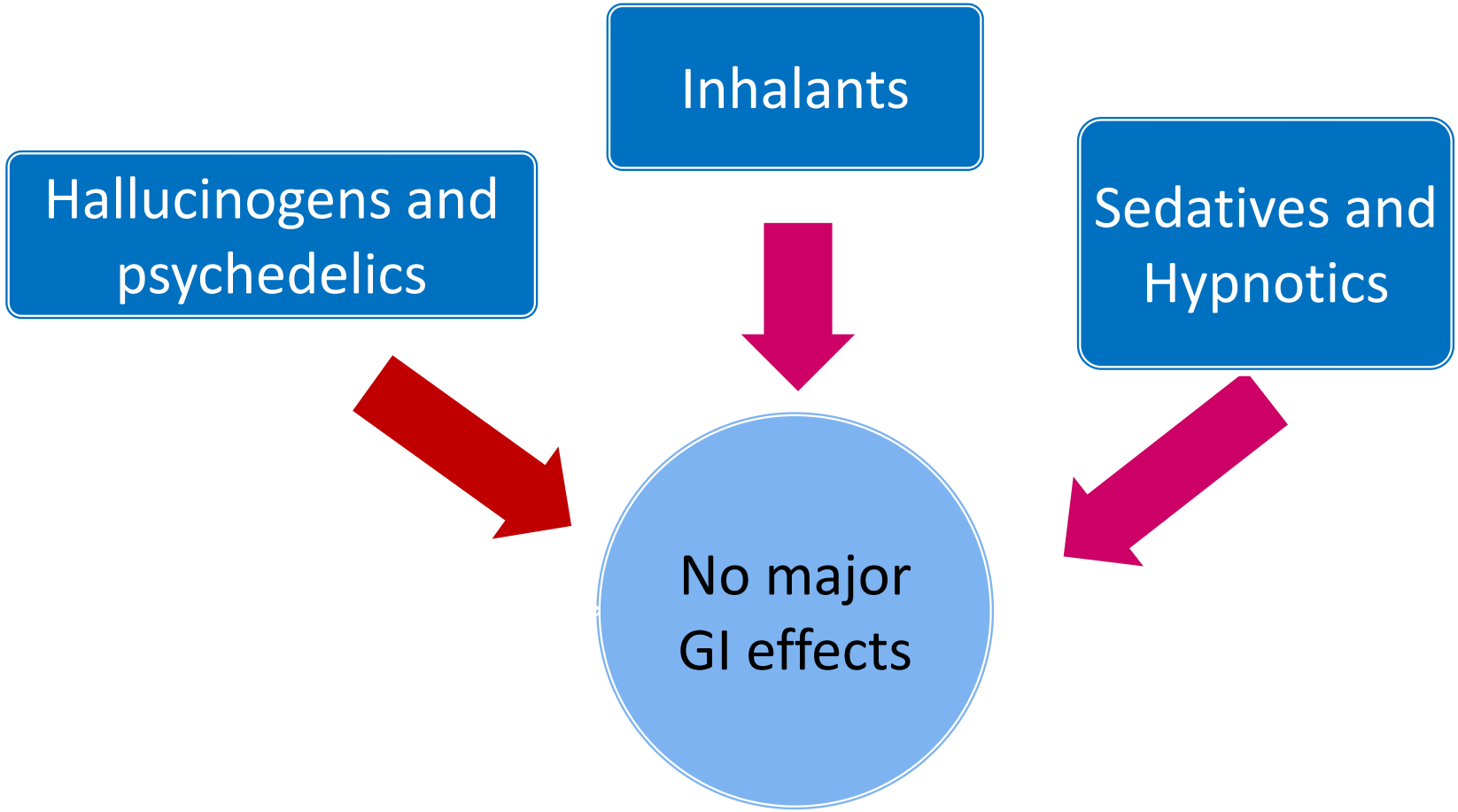
Cannabis

- Cannabis interacts with the endogenous cannabinoid receptors in the digestive tract, which can result in calming spasms, assuaging pain, and improving motility.
- Cannabis has also been shown to have anti-inflammatory properties.
- Anecdotal evidence in patients with IBS, Crohn's disease and other painful GI disorders that cannabis eases cramping and helps modulate diarrhea, constipation and acid reflux.
- POTENTIAL FOR THERAPEUTIC USE!!!

- Adverse effects – few.
- Usually delay in gastric emptying.
- Nausea, acid reflux.
- Munchies and rushes… ?hunger pangs and flushing.
- Constipation.
- Visceral hyperalgesia.
- Electrolyte disturbances.

Amphetamines

- Principally affect the motility. Reduce gastric emptying, and gut transit.
- Anorectic.
- Hyperthermia and dehydration.
- Cramps, colic and diarrhoea.
- IBS like symptoms.


Inhalants
Hallucinogens and psychedelics
Sedatives and Hypnotics
No major GI effects

Smoking and Coffee

- Peptic ulcers.
- Upper GI cancers.
- GI bleed.
- IBS.
- Reflux and GERD.

Thank You

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