# WELCOME TO
# AK LIVER DISEASE ECHO

ALASKA NATIVE TRIBAL HEALTH CONSORTIUM

NPAIHB
Indian Leadership for Indian Health

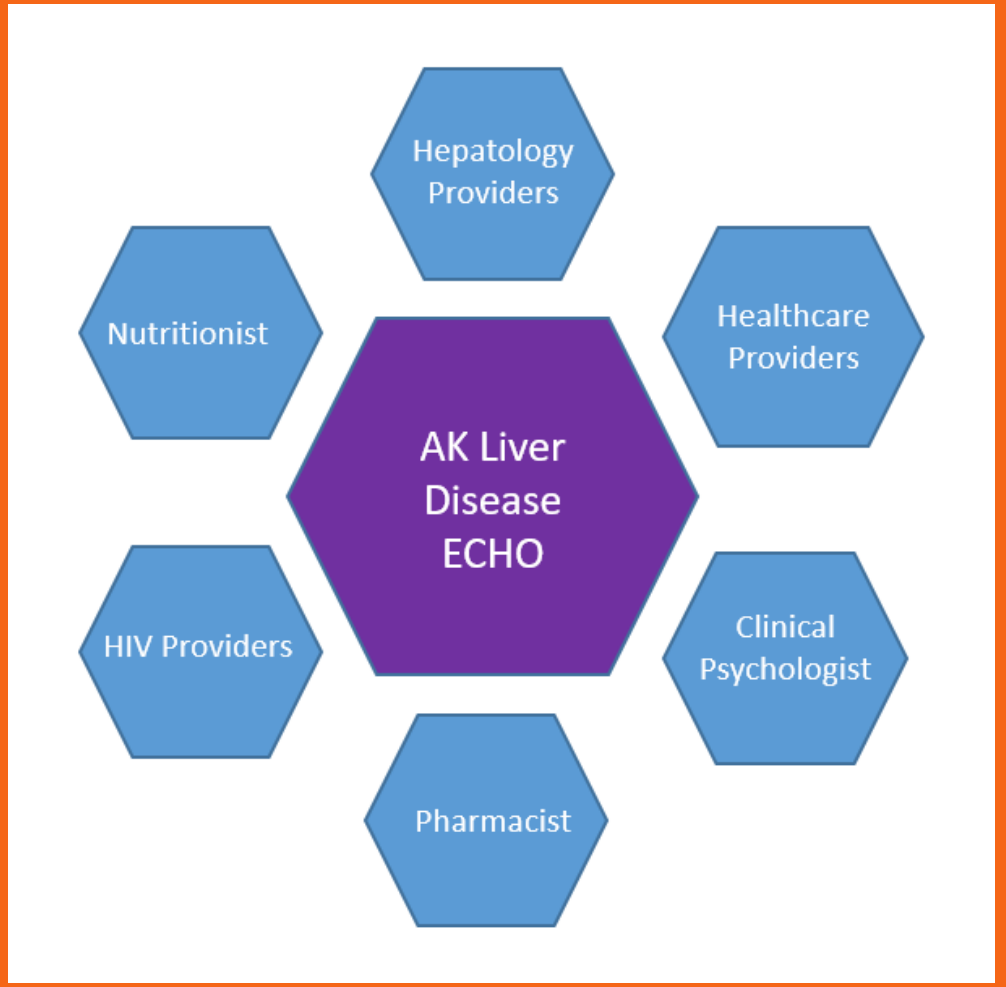
This project is supported by a grant from the Northwest Portland Area Indian Health Board and funding is provided from the HHS Secretary's Minority HIV/AIDS Fund.

# WHAT WE DO

- Didactic Presentations pertaining to ECHO topics
- We're accepting **case presentations and questions pertaining to:**
  - Elevated Liver Function Tests
  - Cirrhosis
  - Managing Complications of Decompensated Cirrhosis – Ascites, encephalopathy, esophageal varices
  - Alcohol-related liver disease, including Alcohol Hepatitis
  - Autoimmune liver disease – Autoimmune Hepatitis, Primary Biliary Cholangitis, Overlap
  - Nonalcoholic fatty liver disease/Nonalcoholic steatohepatitis
  - Hepatocellular carcinoma
- Provide Expert Panelists

# CONSULTANT TEAM

- Brian McMahon, MD Hepatologist
- Youssef Barbour, MD Hepatologist
- Lisa Townshend, ANP Hepatology Provider
- Annette Hewitt, ANP Hepatology Provider
- Leah Besh, PA-C HIV/Hepatology Provider
- Anne Fleetwood, MS, RDN, NDN
- Brittany Keener, PharmD, MPH, BCPS
- Lucia Neander, PhD Clinical Psychologist



# Welcome to Alaska Liver Disease ECHO

**Approved Provider Statements:**
Alaska Native Tribal Health Consortium (ANTHC) is accredited by the Washington State Medical Association to provide continuing medical education for physicians.
ANTHC is approved as a provider of nursing continuing professional development by the Montana Nurses Association, an accredited approver with distinction by the American Nurses Credentialing Center's Commission on Accreditation.
The Alaska Pharmacists Association (AKPhA) in cooperation with ANTHC is accredited by the Accreditation Council for Pharmacy Education as a provider of continuing pharmacy education.

**Contact Hours:**
ANTHC designates this live activity for a maximum 12 AMA PRA Category 1 Credit(s) ™ for the entire series. Physicians should claim only the credit commensurate with the extent of their participation in the activity.
ANTHC designates this activity as meeting the criteria for one nursing contact hour credit for each hour of participation up to a maximum 12 hour(s), including 3 total pharmacotherapeutics (Rx) contact hours for the entire series.
To receive CPE credit, participants must complete an Evaluation/Attendance Form for each session attended. You will be required to enter your NABP e-profile ID number, & birthdate (mm/dd). CPE credit will be posted to the online CPE Monitor system within 60 days after completion of each activity. No credit will be reported to CPE Monitor for CEs that do not have a completed evaluation. There is no charge to process CPE credit for ANTHC employees and AKPhA members, but a fee may apply to participants not affiliated with either organization.

**Conflict of Interest Disclosures:**
Youssef Barbour, MD & Lisa Townshend-Bulson, APRN / faculty for this educational event, are primary investigators in an ANTHC sponsored hepatitis C study funded in part by Gilead Sciences; Anne Fleetwood, faculty for this educational event, is a contractor with Tandem Diabetes Care. All of the relevant financial relationships listed for these individuals have been mitigated.

**Requirements for Successful Completion:**
To receive CE credit please make sure you have actively engaged in the entire activity, your attendance is recorded by the facilitator, and complete the course evaluation form found here: https://forms.gle/R8vibUZgMbRcoScw9.

For more information contact
jlfielder@anthc.org or (907) 729-1387

ALASKA NATIVE TRIBAL HEALTH CONSORTIUM

# Ascites Management

Secondary to Portal HTN.

# No conflict of interests to disclose

Youssef Barbour M.D

# Pre test

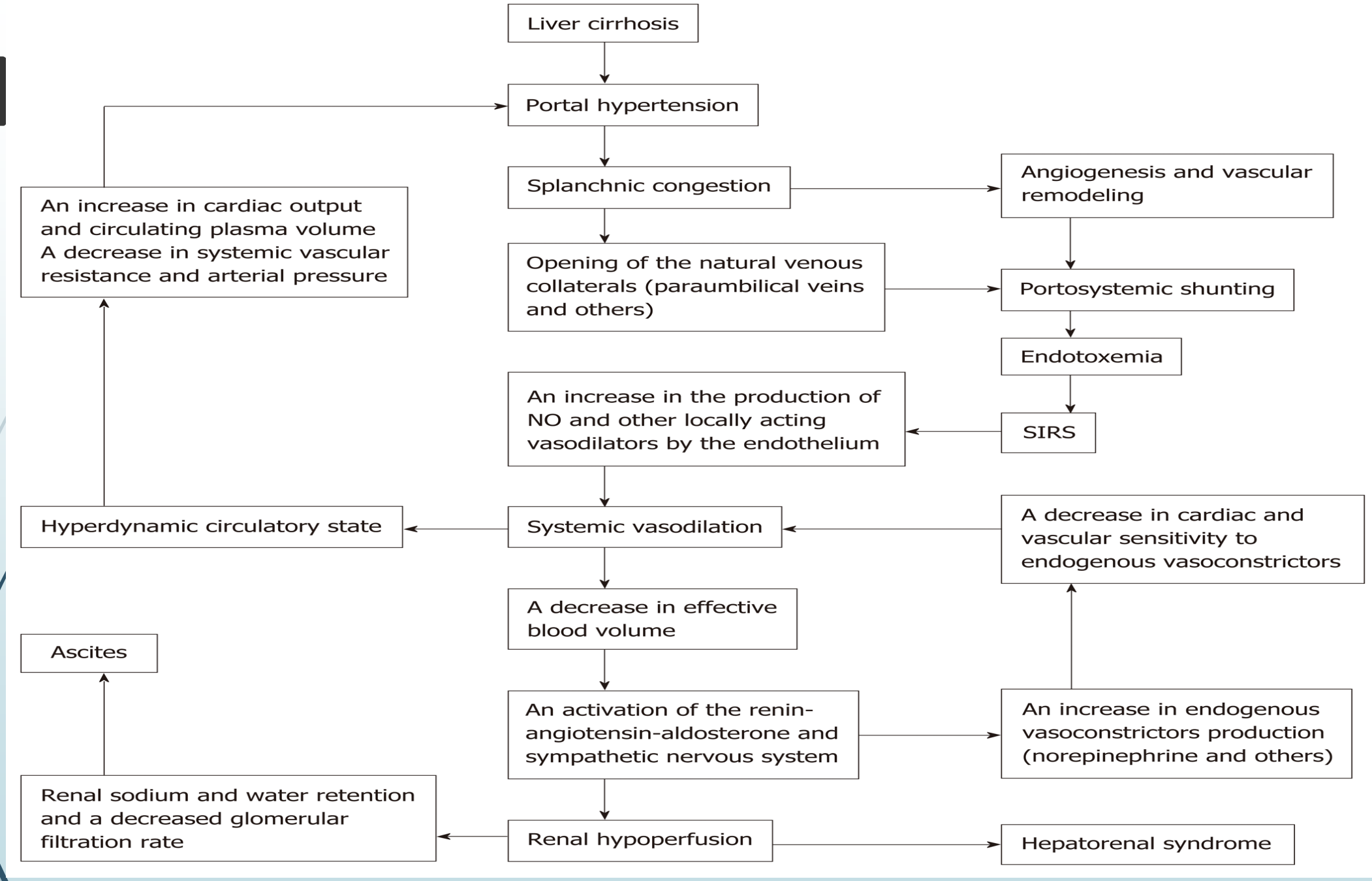
- The preferred monotherapy diuretic to manage Ascites is:
- 1- Furosemide
- 2- Torsemide
- 3- Spironolactone
- 4- Metolazone

# Ascites

- Occurs in about 60% of patients with compensated liver cirrhosis within 10 years after establishing the diagnosis
- Mortality reaches 40% within a year and 50% within 2 year
- In the case of refractory ascites, median survival does not exceed 6 months due to the development of more related complications

# Pathophysiology

- At present, the leading theory of ascites formation is the hypothesis of peripheral arterial vasodilation, the reasons for which include systemic inflammatory response (SIRS)


Liver cirrhosis
Portal hypertension
Splanchnic congestion
Angiogenesis and vascular remodeling
An increase in cardiac output and circulating plasma volume
A decrease in systemic vascular resistance and arterial pressure
Opening of the natural venous collaterals (paraumbilical veins and others)
Portosystemic shunting
Endotoxemia
An increase in the production of NO and other locally acting vasodilators by the endothelium
SIRS
Hyperdynamic circulatory state
Systemic vasodilation
A decrease in cardiac and vascular sensitivity to endogenous vasoconstrictors
A decrease in effective blood volume
Ascites
An activation of the renin-angiotensin-aldosterone and sympathetic nervous system
An increase in endogenous vasoconstrictors production (norepinephrine and others)
Renal sodium and water retention and a decreased glomerular filtration rate
Renal hypoperfusion
Hepatorenal syndrome

# Classifications of cirrhotic ascites

## Uncomplicated

- **No** infection of HRS
- **Grade 1**: detected by US
- **Grade 2**: moderate, detected by physical exam
- **Grade 3**: massive tense ascites

## Refractory

- Does not recede to at least grade 1 with the use of diuretic treatment and dietary sodium restriction
- **Or**; early recurrence of which after large volume paracentesis cannot be satisfactory prevented by medical therapy
- *It has 2 subtypes*: **diuretic-resistant** and **diuretic-intractable**

# Ascetic fluid analysis

## Portal HTN

- Calculating **SAAG**:
- >1.1 portal HTN or cardiomyopathy.
- **Total protein** in the ascetic fluids: <2.5 indicates portal HTN origin, >2.5 indicates cardiac origin

## Differential Diagnosis

- **Chylous ascites**: cloudy color, TGs>200 mg/dl, lymphocytes >500/ml, cholesterol gradient <1, SAAG>1.1
- **Malignant ascites**: C-RP high in ascetic fluids and blood serum, high insulin-like growth factor-1, cholesterol>45 mg/dl, and CEA; in addition to cytology
- **TB**: Adenosine Deaminase

# Spontaneous Bacterial Peritonitis

- **Neutrophils count** > 250 cells/mm is the diagnostic criterion for it. (with or without positive culture)

- **<u>SBP prophylaxis</u>**:

- A- ascites with **H/O SBP**

- B- **ascetic total protein < 1.5 g/dl <u>AND</u> severely impaired liver (CTP class C, bilirubin >3) <u>AND</u> renal (creatinine>1.2, BUN >25, or Na <130) function**

# Treatment of Ascites

- **Management of uncomplicated ascites** (not accompanied by infection or hepatorenal syndrome)
- **Management of refractory ascites**

# Management of uncomplicated ascites

- **Grade I ascites**: no need for diuretics and low Na diet
- **Grade II ascites**: Na restriction (4.6-6.9 g/day), greater restrictions are undesirable, as the deterioration in food taste can lead to anorexia, diuretics also can be used, *if monotherapy is attempted*; use aldosterone antagonists (i.e.; spironolactone) instead of loop diuretics. Can be used up to 100-200 mg spironolactone daily, then, in the absence of an effects, furosemide can be added. *Alternatively, combined use can be attempted from the beginning* (doses up to 400 mg spironolactone and 160 mg of furosemide
- *Should target weight loss while on diuretics not to exceed 500 gm (1kg if there is peripheral edema)daily*, goal to reach ascites grade I, diuretics then can be titrated off

# Grade III uncomplicated ascites "tense ascites"

- Recommendations to **start with Large Volume Paracentesis (LVP), then diuretics and salt restrictions.**
- **Usual amount 5-6 liters**
- The frequency of severe intra-abdominal bleeding during LVP is <1% (use of FFP or platelets are reserved for certain cases like severe liver impairment, or severe thrombocytopenia)
- **The most dangerous consequence of LVP is paracentesis-induced circulatory dysfunction (PICD)**, *characterized by* severe hemodynamic disturbance which is accompanied by increased cardiac output, decreased central venous pressure, and peripheral vascular resistance reduction.
- **PICD is associated with** *frequent recurrence of ascites*, *dilutional hyponatremia*, *hepatorenal syndrome development* and *high mortality*
- **Albumin infusion** of the amount 8 gm/ 1 L removed ascetic fluid may prevent this complication

# Treatment of refractory ascites

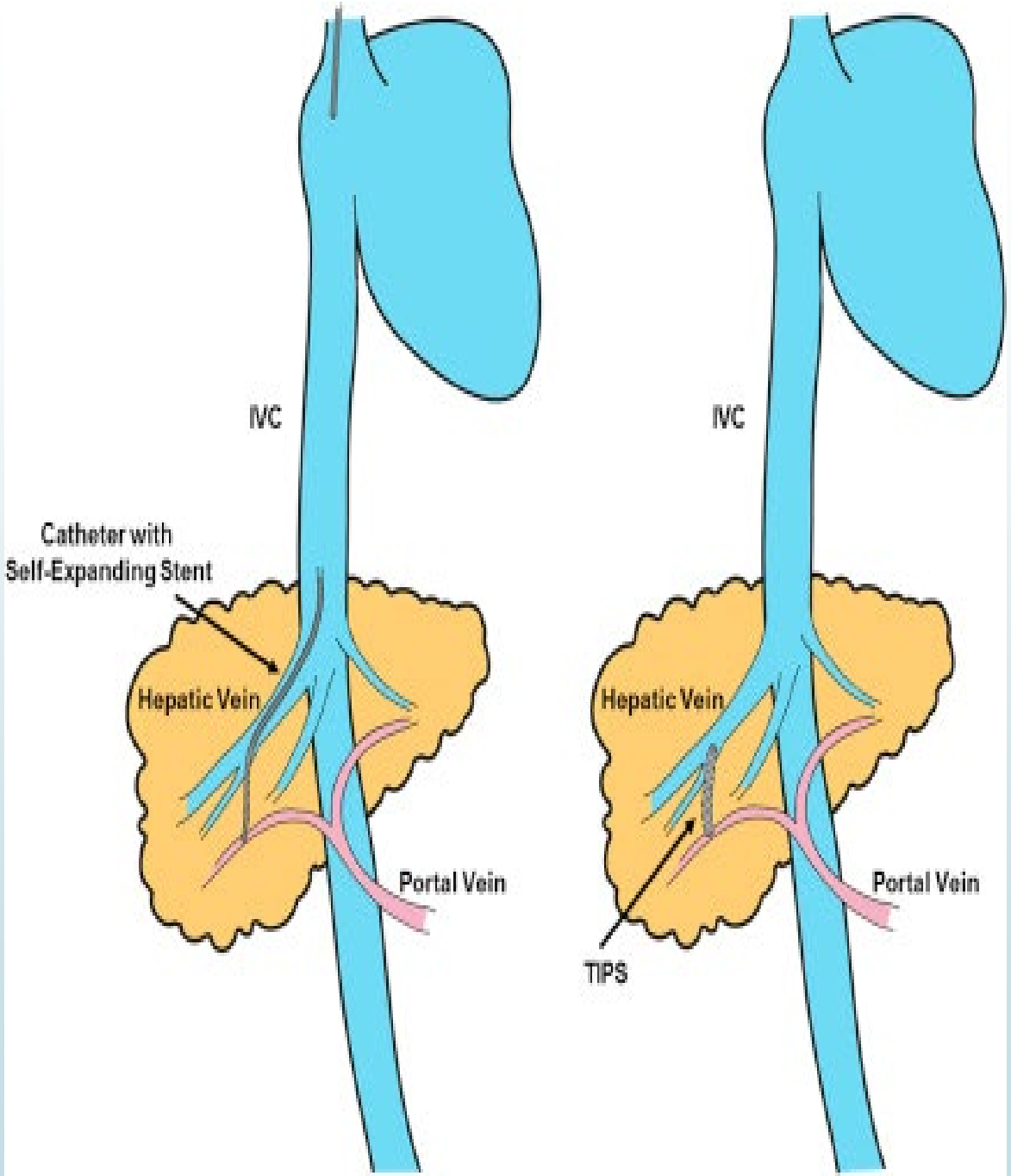
- **The first-line therapeutic intervention is LVP repeated every 2-3 weeks in combination with albumin infusions**.
- **Make sure** loop diuretics is not being used as monotherapy
- **Make sure** diuretics used are not causing rapid weight loss (volume loss) as this hypovolemia can lead to pre-renal azotemia
- **Midodrine** ( alph1 agonist) can be added to boost the MAP to help renal perfusion, and boost the effects of diuretics.
- **If hyponatremia develops (<125) diuretics need to be stopped, and limit fluid intake to 1 L/day**
- Hyponatremia can be corrected by **tolvaptan**, selective vasopressin V2 receptor antagonist (inhibiting ADH) but with high hepatotoxicity rate (up to 20%), so risk vs. benefit should be assessed before its use, with close monitoring while in use (no need for water restriction if tolvaptan is being used)
- Consider **liver transplant referral**, consider **TIPS**

# TIPS (Transjugular Intrahepatic Portosystemic Shunting)

Improve ascites in 50-70% of cases.

**Contraindications include**:

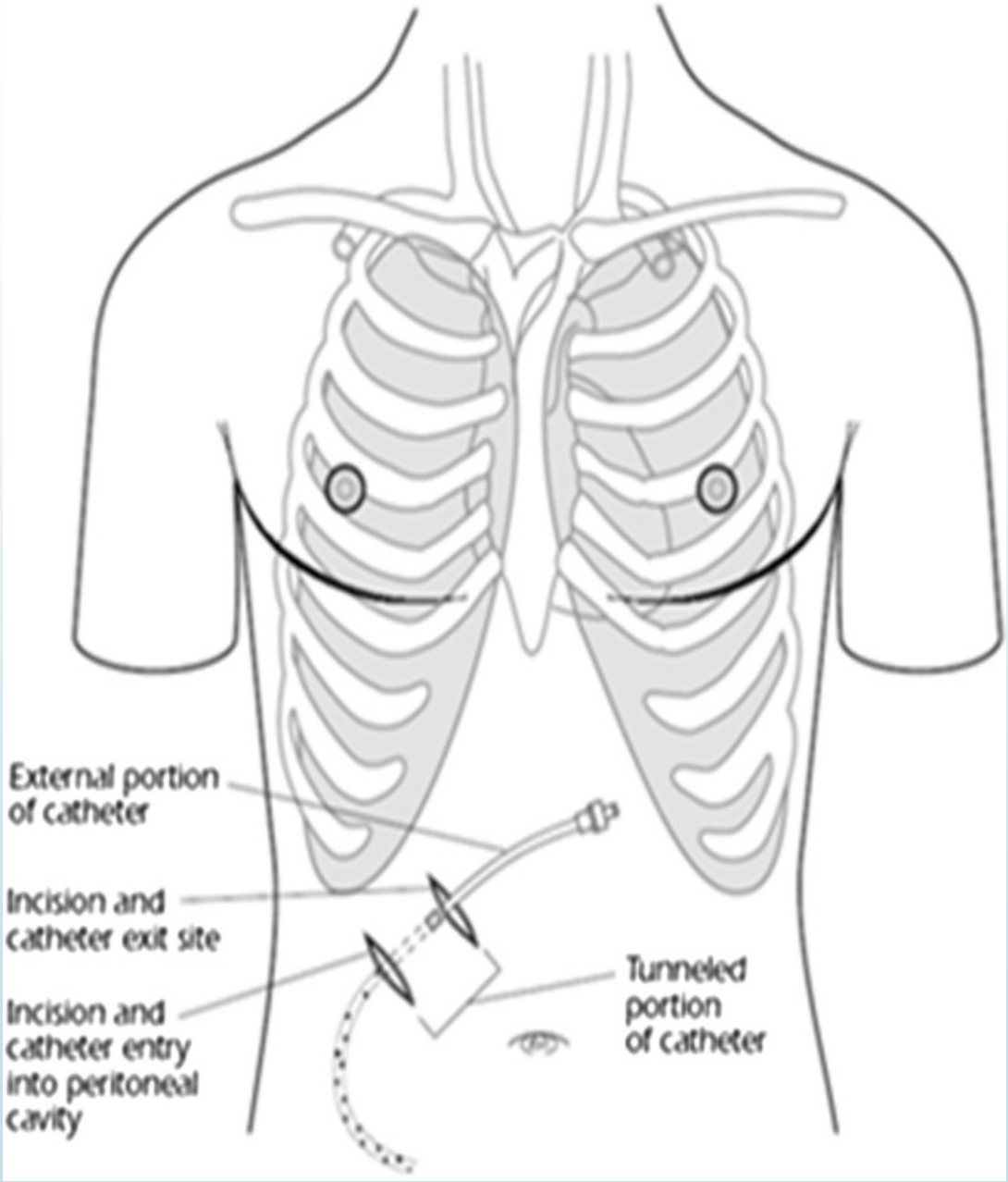
MELD>23, Bilirubin>4, diastolic dysfunction, recurrent encephalopathy



# Other Options

**Permanent Pleurx Tunneled Peritoneal Catheter, is used in malignant ascites. Needs prospective RCT**

There are shunts between abdominal cavity and bladder, and peritoneovenous shunts, those has significant sides effects reported.



## CIRRHOSIS CARE DOCUMENT

https://anthc.org/wp-content/uploads/2020/12/Care-Cirrhosis.pdf

Ascites – Perform diagnostic paracentesis for new onset or if patient admitted to hospital or has a change in clinical status. Sodium restricted diet 2000mg/day. If serum sodium is ≥135mEq treat with a **single morning dose** of spironolactone and furosemide, pending BP tolerability. Consider fluid restriction and increasing furosemide dose to raise serum sodium.

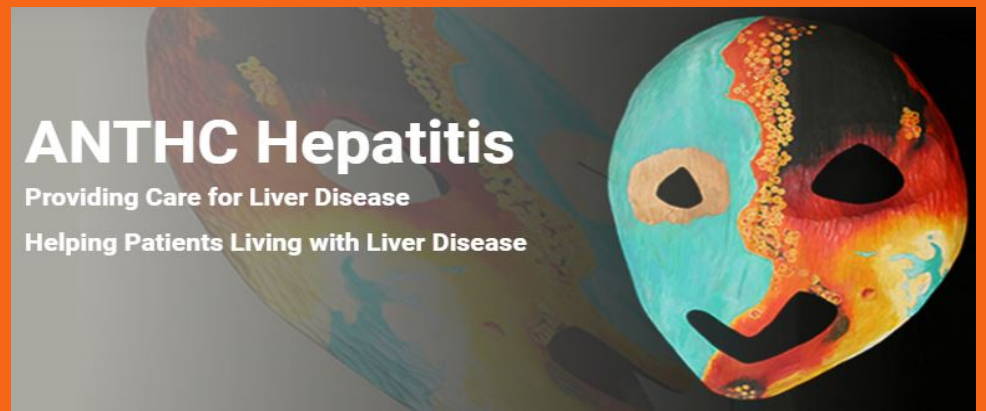
| **Drug – Give Both Drugs Below as Single A.M. Doses** | **Initial dose** | **Adjust Dose Based on BP Tolerability and to Maintain Na Level > 135mEq** |
|---|---|---|
| Spironolactone | 100mg | Escalate dose every 3-5 days as needed. Decrease if Na <135mEq and stop if Na <130mEq. |
| Furosemide | 40mg daily | Monitor KCL and Sodium. Can increase to raise sodium level. |

# LIVER DISEASE ECHO SCHEDULE AT A GLANCE

- June 17 Motivational Interviewing – Lucia Neander, PhD
- July 15 Portal Hypertension and Varices – Youssef Barbour, MD
- August 19 Drug Induced Liver Injury – Brittney Keener, MPH, BCPS
- September 16 HCC Surveillance – Brian McMahon, MD
- October 21 Treatment of Acute Alcohol Hepatitis – Brian McMahon, MD

# ADDITIONAL LEARNING OPPORTUNITIES

- AK ID ECHO:  HCV, HIV, PrEP, STIs
  - The 2nd Tuesday of every month from 12:00-1:00PM Alaska Standard Time
    - 1CE/CME offered per session
  - anthc.org/project-echo/hcv-hiv-prep-stis-echo
- LiverConnect Webinar Program
  - Second Tuesday of every month 8:00-9:00AM Alaska Standard Time
    - Full Hour didactic topics on Liver Disease and related topics 1CE/CME offered
  - anthc.org/what-we-do/clinical-and-research-services/hep/liverconnect/



# AK LIVER DISEASE ECHO -TEAM CONTACTS

- Lisa Townshend-Bulson, MSN, FNP-C, Program Manager, ltownshend@anthc.org
- Danielle Varney, Program Coordinator, dvarney@anthc.org
- Wileina Rhodes, RN  Nurse CE Coordinator, wsrhodes@anthc.org
- Annette Hewitt, FNP-C Pharmacology Content Reviewer, amhewitt@anthc.org
- Cindy Decker, RN  Liver Disease ECHO Nurse Case Manager, cadecker@anthc.org
- ANTHC Liver Disease and Hepatitis Program: 907-729-1560
- Northwest Portland Area Indian Health Board
  - David Stephens: Director Indian Country ECHO, dstephens@npaihb.org
  - Jessica Leston: Clinical Programs Director, jleston@npaihb.org

# Thank you

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