腸胃道出血

Gastrointestinal Tract Bleeding

個案介紹

詹先生 44 歲，因腸胃道出血由急診室轉到加護病房治療，急診室的記載陳述他於一個星期即因參加新年舞會兒喝了很多酒後，一直有上腹痛情形，而在門診治療。他主訴舞會後覺得上腹部非常痛，強烈噁心想吐感長達 48 小時，他被診斷為胃炎，予止吐劑和制酸劑回家服用，並被勸告要停止喝酒，雖然它回到日用品貿易商的工作，但自從舞會後都沒覺得比較舒服，除了處方藥外，由於上腹痛自行額外服用 aspirin 2-3 次/天，每次兩顆，此次因為噁心及兩次嘔吐出大量暗褐色水狀物且極度虛弱而到急診室，他在突然站起或坐起時會有眩暈感。

在 6pm 住院時的 vital sign 及實驗室檢驗值如下：

體溫 37.9℃（耳溫）　　心跳 102 次/ 分　　呼吸 20 次/分

血壓　躺　96/60mmhg　　站 84/50mmhg

血紅素 12.5g/dl　　血比容 40%　　白血球 1200/mm^3

在急診室，詹先生置入 14 號鼻胃管，引流出 350ml 暗褐色、有潛血反應和咖啡渣狀液體流出，以生理食鹽水 500ml 灌洗胃後引流液終於變為清澈。入 ICU 後，詹先生的處方為：

1. 每小時監測心電圖、Vital signs、攝入和輸出量
2. 維持臥床休息
3. 鼻胃管以低壓間歇裝置抽吸，如有主動出血情形則通知醫師，並以生理時鹽水每兩小時或需要時沖洗直到引流液清澈為止。
4. 除口服藥外 NPO。
5. 口服給藥，給予時將鼻胃管夾住 30 分鐘
6. Magnesium hydroxide（Mylanta）30 ml q4h PO.
7. Cimetidine（Tagamet）300 mg q6h PO.
8. Promethazine（Phenergan）25 mg IV q6h prn for nausea.
9. 5%dextrose in lactated Ringer's solution(D5LR）IVD, 100 ml/hr.
10. Esophagogastroduodenoscopy（EGD）at 7am 在腸胃科檢查室
11. 晚上 9 點測血紅素、血比容
12. 在早上測全血球計數、血小板、部分血栓形成質時間（PTT）凝血酶原時間部分（PT）生化 12 項、電解質、小便分析

傍晚詹先生沒有特別的變化，他的病歷記錄為：

	HR	RR	BP	其他測量值
7PM	112	16	104/64	
8PM	115	18	100/60	BT:37.9
9PM	120	20	98/60	
10PM	120	20	100/60	Hb:12.3g/dl、Hct：36%
11PM	120	20	96/54	sleep
Midnight	112	20	92/50	BT:36.7（耳溫） 120ml 琥珀色小便

在午夜時鼻胃管因給藥夾起，當鼻胃管夾在 12:30AM 打開時，300ml 血色的引流液流出，他變的不安和焦慮，皮膚蒼白且濕冷，予 O2 Nasal cannula 4L/min，在 12：30AM 時血壓：84/50mmHg、HR：126 次/分、RR：28 次/分

通知醫師到場處理，處置如下：

1.以 N/S 沖洗鼻胃管直到清澈為止。

2.立即測量 Hb 和 Hct，並回報結果。

3. 血型和交叉試驗 packed red blood cells（PRBCs）2 units；另比對和篩檢

4. 鎖骨下放置三腔靜脈點滴管路。

5.每小時測量 CVP 值，假使少於 10mmHg，給予 N/S 500ml 靜注輸液快速給予，需要時可重複給予。

6.放置導尿管並測量每小時小便量。

7.通知腸胃科醫師和外科醫師。

詹先生導尿管放置無小便流出，1 am 時檢驗值如下 ：

CVP:8mmHg　　Hgb:9.8g/dl　　Hct:24%

1:30 am 快速給予 500 ml 水分後，CVP12mmHg，小便量 30ml/小時。D5LR 50ml/hr 由周邊管路注射，NS 150ml/hr 由中央管路注射，2 am 時輸 2U PRBCs。

之後其 Vital signs 和其他測量值如下：

時間	心跳（bpm）	呼吸（次/分）	血壓（mmHg）	CVP（mmHg）	小便量	其他臨床資料
2 am	120	26	84/50	10	30	
3 am	116	24	90/58	12	75	
4 am	112	20	94/26	12	90	
5 am	110	18	96/64	12	94	
6 am	110	18	98/62	13	80	
7 am	104	18	98/64	12	68	Hb10.1g/dl, Hct 28%

在早上 7:10 詹先生被送到腸胃科檢查室作食道胃腸鏡檢，腸胃科醫師發現有瀰漫性胃炎和一個 2 公分的十二指腸潰瘍，沒看到血塊，也無主動性出血情形，胃壁切片檢體呈幽門螺旋桿菌試驗呈陽性反應，也做了培養及敏感試驗。

詹先生開始接受幽門螺旋桿菌藥物治療、並服用 histamine-受體對抗劑和制酸劑，醫師決定採保守療法，詹先生病情也逐漸好轉，24 小時後就轉到普通病房，三天後出院。

討論題

- What is the incidence of and mortality with acute GI tract bleeding?
- Identify common causes of GI tract bleeding and list predisposing factors specific to Mr. Jones.
- Discriminate between the characteristics of upper and lower GI tract bleeding.
- What complication did Mr. Jones experience
- Which factors determine whether blood products will administered to a patient with GI tract bleeding?
- Mr. Jones' Hgb and Hct values dropped dramatically from admission to 7 am. Discuss the drop in Hgb and Hct values in relation to Mr. Jones' blood loss.

討論題（續）

- If a patient continues to have active bleeding from the GI tract despite conservative management, what medical procedures might be implemented?
- Identify pharmacologic therapy commonly used in the treatment of GI tract bleeding.
- What are the indications and types of surgical procedures for upper GI tract bleeding?
- Identify six nursing diagnoses appropriate for Mr. Jones.
- What is the incidence of *Helicobacter pylori* infection in gastritis and duodenal ulcers?
- What is the treatment of choice for *Helicobacter pylori* infection?

Incidence and mortality with acute GI bleeding

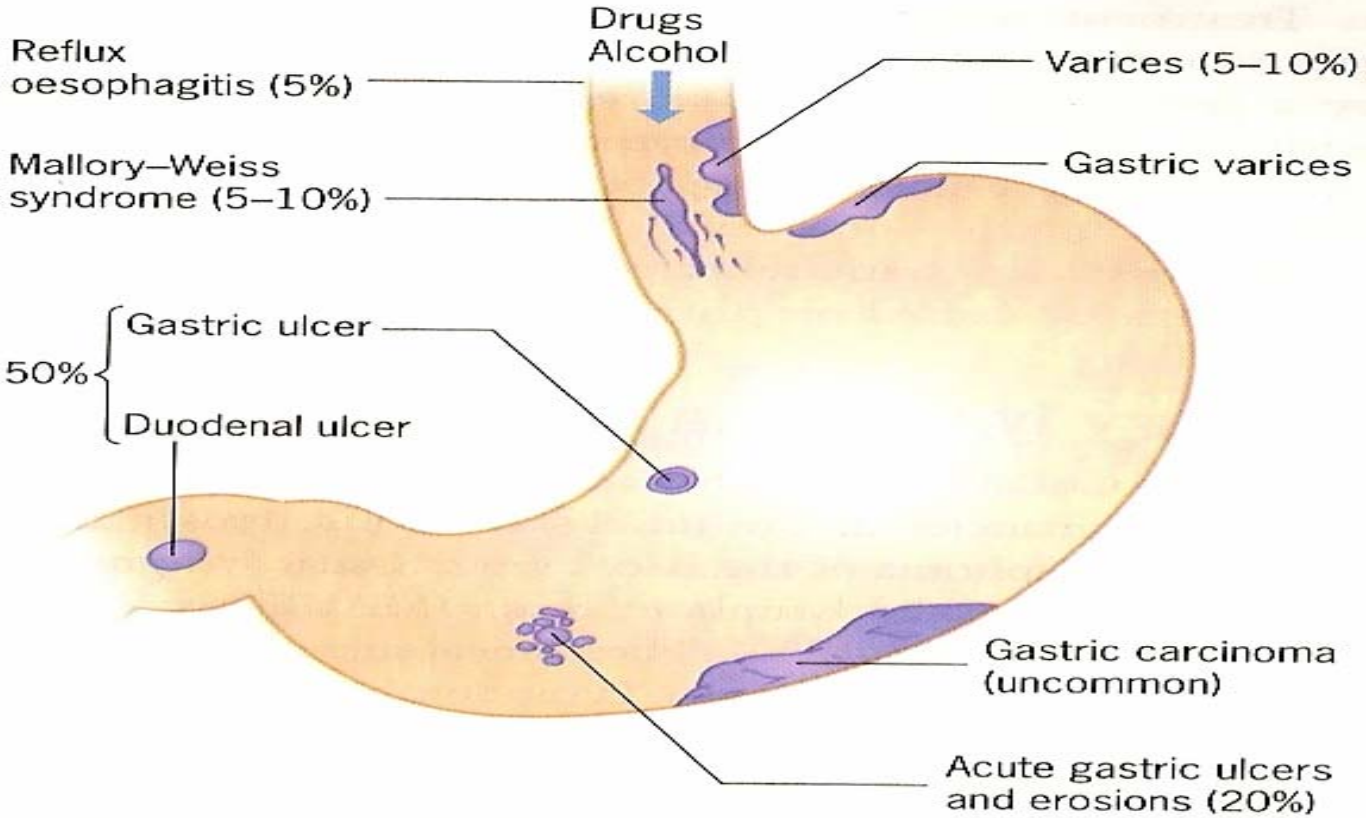
- Overall mortality rate: 10%
  - Rupture of esophgeal varices (mortality rate 40~70%)
- Increases in
  - Age >60
  - Shock on admission
  - Rebleeding within 72 hours
  - 經灌洗引流液無法轉清

Common cause

- Upper GI Bleeding
  - 大部分位於食道下1/3至12指腸球莖間。
    - Peptic ulcer
    - Telangiectasia(毛細管擴張)
    - Stress ulcer
    - Esophageal ulcer
    - Mallory-Weiss syndrome(食道壁胃壁線性裂傷)
    - Esophatitis
- Lower GI Bleeding
  - 大部分位於升結腸以下腸道。
    - Colon angioma, diverticula, internal hemorrhoids

GI bleeding的病理機轉及相關因素

- 長期腸胃道黏膜發炎及潰瘍
  - 長期酗酒、劇烈嘔吐易致Mallory-Weiss tears。
  - Aspirin及NSAID服用發生潰瘍機率是5-25%，為不使用NSAID患者的2倍。
  - 曾潰瘍者，有40-50%再出血率。
- 腸道靜脈曲張，腹壓上升，腸道靜脈破裂
  - 門靜脈壓>15mmHg易致食道靜脈曲張出血。
  - 慢性便祕，內痔出血。
- 急性腸胃道組織灌流不足
  - 壓力性腸道潰瘍，休克、敗血症。



Other uncommon causes
Hereditary telangiectasia (Osler–Weber–Rendu syndrome)
Pseudoxanthoma elasticum
Blood dyscrasias
Dieulafoy gastric vascular abnormality
Portal gastropathy

Fig 4.16
Causes of upper gastrointestinal haemorrhage. The approximate frequency is also given

詹先生之出血因素

- 喝酒
- 抽菸
- 服用Aspirin
- 工作壓力

急性腸胃道出血的表現

- 吐血(hematemesis)
  - 吐鮮血或coffee ground ，表示是12指腸韌帶近端的出血，往往大於1000ml。
- 黑便、柏油便 (melena, tarry stool)
  - 升結腸遠端的出血100ml以上。解黑便出血量通常在500ml以下
  - 假性黑便包括食用鐵劑、甘草、藍莓、活性碳。
- 血便(hematochezia)
  - 12指腸韌帶之後出血
  - >1000ml以上的上腸胃道出血吐血血便都有。

急性腸胃出血的加護策略

- 整體性考量
  - 立即評估
    - 失血量和血流動力的穩定度。
    - 實驗室評估：Hb,Hct,PT, APTT,lactate
  - 即刻處置
    - 增加血管內血流量
    - 矯正凝血病變。
- 病史
  - 何時開始出血、出血顏色、原有疾病、特殊服用藥物。
- 身體檢查
  - 排便顏色、NG抽吸腸胃液顏色量。

失血量的立即評估

- 血壓下降：
  - 原來正常血壓者，收縮壓低於90-100mmHg
  - 原有高血壓者，血壓下降20-30mmHg。
- 姿位性低血壓
  - 體位變更(平躺至坐起)收縮壓降低10mmHg以上，或脈搏增加每分鐘20跳以上，表示體內血循環量減少將近1/4。
- 蒼白，四肢發冷，大量冷汗，呼吸急促。
- 尿量每小時不足25-30cc，表示腎血流減少。

Classification of Volume Loss

分級	失血量	S/S
1	15%	HR↑ RR ↑ BP↓但在正常範圍, Syncope, Nausea
2	20~25%	HR>110, RR>25, 微血管回填> 5 sec. Orthostatic Hypotension, 坐起時BP drop >10 mmHg
3	30~40%	HR>110, RR>25, 微血管回填> 5 sec 平躺低血壓 SBP <100mmHg, Oliguria
4	>40%	HR>110, RR>25, BP 量不到，微血管回填> 5 sec, Cardiovascular Collapse

詹先生經歷之出血合併症

- Hypovolemia
- Tachycardia
- Tachypnea
- Low CVP
- Orthostatic hypotension
- Low urine output
- Skin pale, cold, restless
- Class III hemorrage (30%~40% blood loss)

GI bleeding 輸血之決定

- Unstable VSs even after 2L of crystalloid fluid replacement
- Hgb< 10 g/dL, Hct < 25%
- Presence of active bleeding

出血與 Hgb 與Hct 值下降的關係

- 出血初時Hgb 與Hct 值無法確實反映血量之流失
  - 血球一起流失，Hgb 與Hct 值維持
  - 細胞內液、組織間隙液轉移，約48~72才達平衡
- 開始輸液的稀釋效果，Hgb 與Hct 值下降
- 輸whole blood, PRBC, Hgb/Hct可上升

- 詹先生之Hgb/Hct
  - 在急診室時Hgb/Hct 12.5/40
  - 入ICU輸D5LR 100ml/hr, Hgb/Hct 12.3/36
  - Challenge NS 500ml Hgb/Hct 9.8/24
  - D5LR 50ml/hr, NS 150 ml/hr, PRBC 2U後, Hgb/Hct 10.1/28

Critical GI bleeding

- 急性腸胃道出血 (GI bleed)病人，併有
  - 凝血障礙(coagulopathy)
  - 低血壓(hypotension)
  - 意識混亂(neurology dysfunction)
  - APACH II score≧15分

需要加護護理，預防進一步的傷害。

Inaget, 2000

即刻處置：增加靜脈回流

- Autotrasfusion maneuvers
  - 重力操作：腿抬高10-45度
  - 腿部血液轉移，增加靜脈回流，增加CO。
- Pneumatic garments
  - 充氣衣(抗休克衣)：
  - 藉由壓迫下肢小靜脈的反壓力設計，增加靜脈回流，用於外傷運送。

液體補充

- 主目標：增加組織氧攝取進行有氧代謝。
  - VO_2=CO X Hgb X 13.6 X (SaO_2-SvO_2)
  - 急性血量流失立刻引起low CO，接著是貧血
- Supporting CO:以增加CO為優先，
  - colloid優於RBC,RBC因血流阻力增加反而減少CO。
  - 增加CO效果上 3份 crystalloid 等於1份Colloid
- Hemoglobin replacement ：
  - 一旦CO得support，開始輸血，補到Hgb 10g/dl
  - 凝血病變者：補FFP。

不同輸液的體液增加效果

	輸入量	TBW	ICF	組織間液	血管容積
D5W	1L	1L	↑800ml	↑ 100ml	↑ 100ml
Lactate Ringer	1L	1L	--	↑ 700ml	↑ 300ml
3%NaCl	1L	1L	↓2.5L	↑ 2400ml	↑ 1100ml
25% Albumin	100ml	100ml	--	漸下降 450ml	↑ 550ml

液體補充量

- 流速=壓力*(導管半徑^4/ 8*黏性*管長)
  - 最大的注射速率決定於catheter diameter的大小，不是血管的大小。要大靜脈是錯誤的觀念
  - 導管長度決定流速阻力，長的cvp比短的cath阻力大滴的慢。
  - 黏度：plasma是1.8，全血是3-4。
- Resuscitation Rules
  - Whole Blood= Volume Deficit
  - Colloid = Volume Deficit
  - Crystalliod = 3 of Volume Deficit
- Normal blood volume

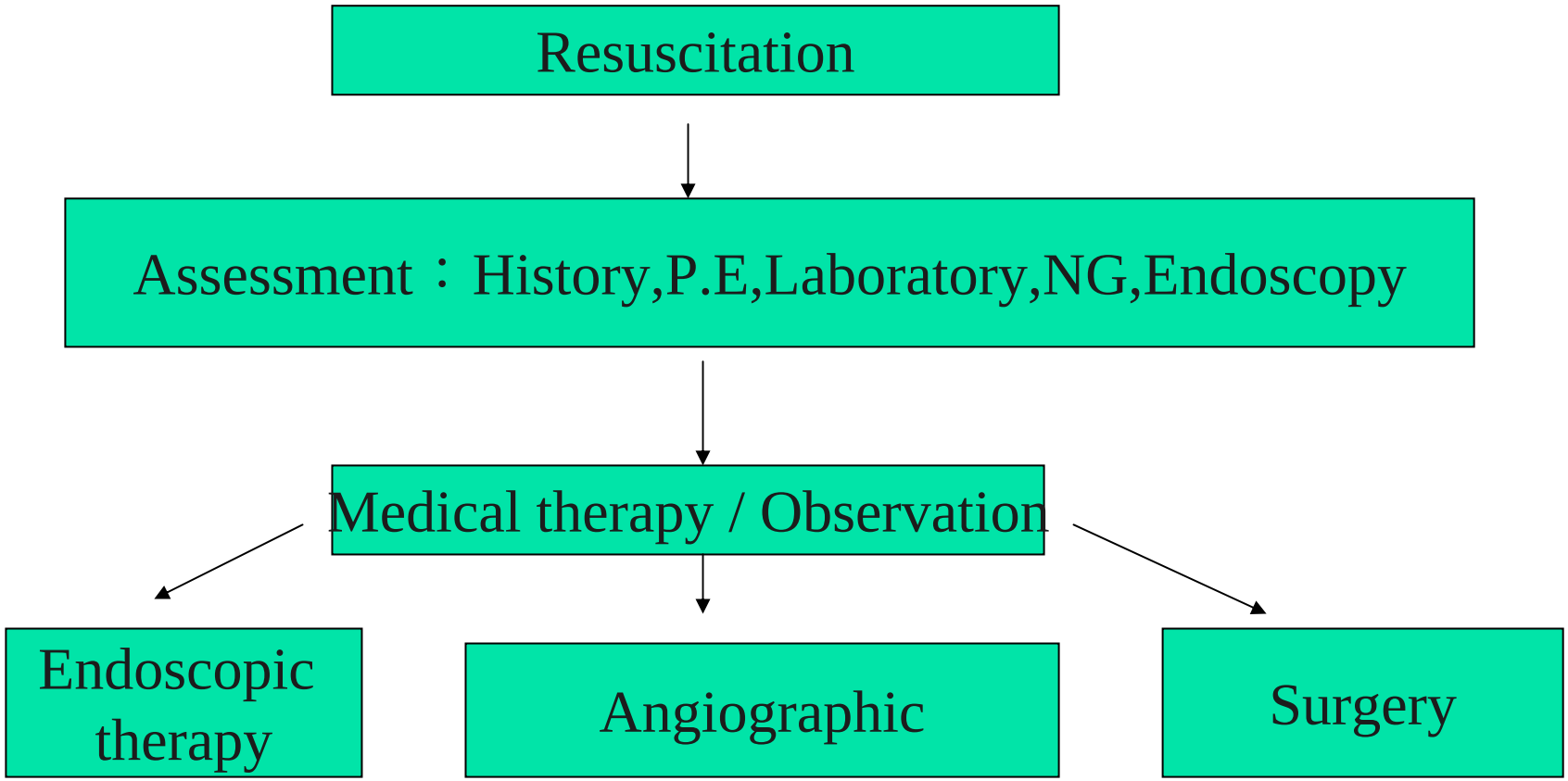
  Male: BV = 70ml/kg

  Females : BV = 60ml/kg

Endpoints

- Goals:
  - MAP>70 mmHg
  - HR: 60~100 bpm
  - CVP=2~6 mmHg (3~8 cmH2O)
  - PCWP=10to 12mmHg
  - U/O >= 0.5 ml/kg/hr
- 以serum lactate監測組織氧合
- 以Hemmatocrit(40%)作為適當水分容積治療結果的指標。

Management of GI Bleeding



Medical intervention

- Ice saline
- Endoscopic hemostasis
  - 燒灼、sclerotic therapy, banding ligation
- Vasopressing infusion
- Octreotide
  - Long acting analog of somatostatin, selective splanchnic vasoconstriction without cardiac complicaiton
- Angiography, transcatheter embolization
- Mechanical temponade
  - S-B tube
- For persistent bleeding: Billroth I, II surgery

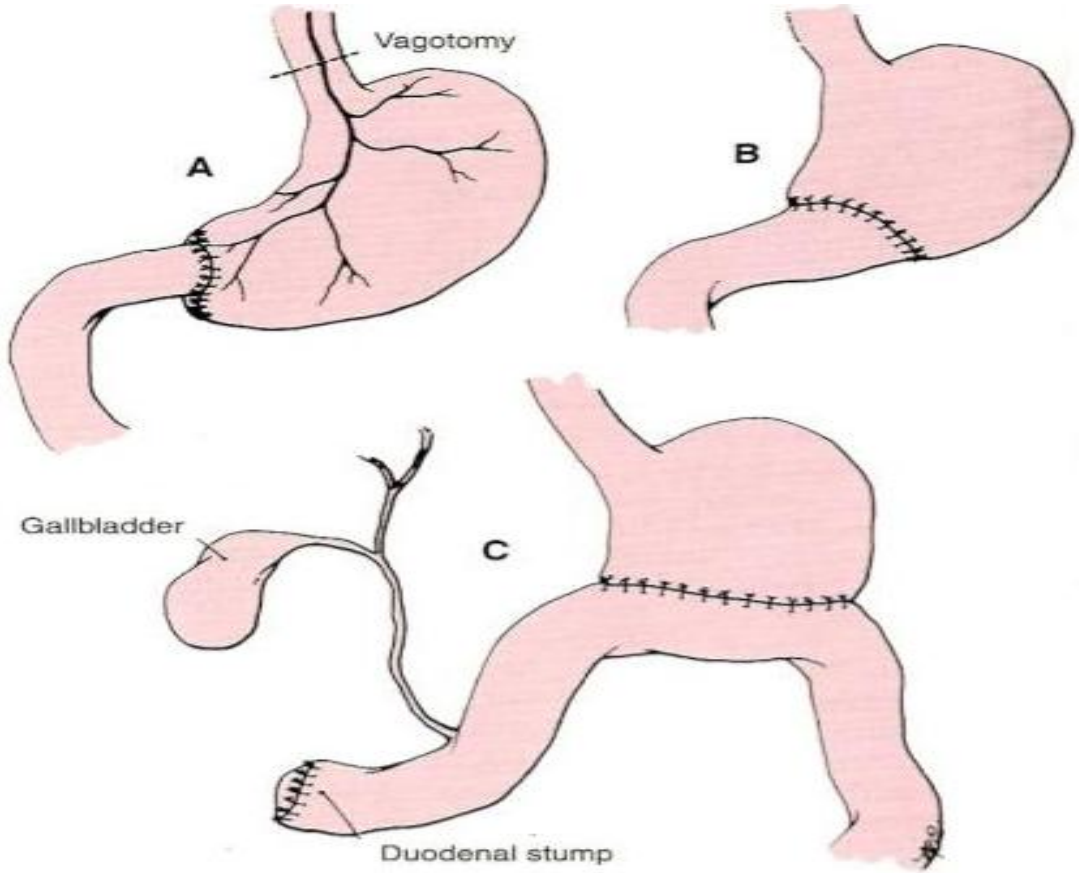


FIG. 24-9 Common surgical procedures for treating peptic ulcers. **A**, Vagotomy plus antrectomy (removal of pyloric antrum). **B**, Billroth I procedure (gastroduodenostomy anastomosis after resection). **C**, Billroth II procedure (gastrojejunostomy anastomosis after resection).

Pharmacologic therapy

- Histamine-receptor antagonist
  - Reduce acid secretion
  - Side effect: headache, dizziness, diarrhea, constipation
- Proton pump inhibitor
  - Omeprazole (Prilosec)
  - 用於等或不能作內視鏡治療時之非動脈噴血
- Antacid

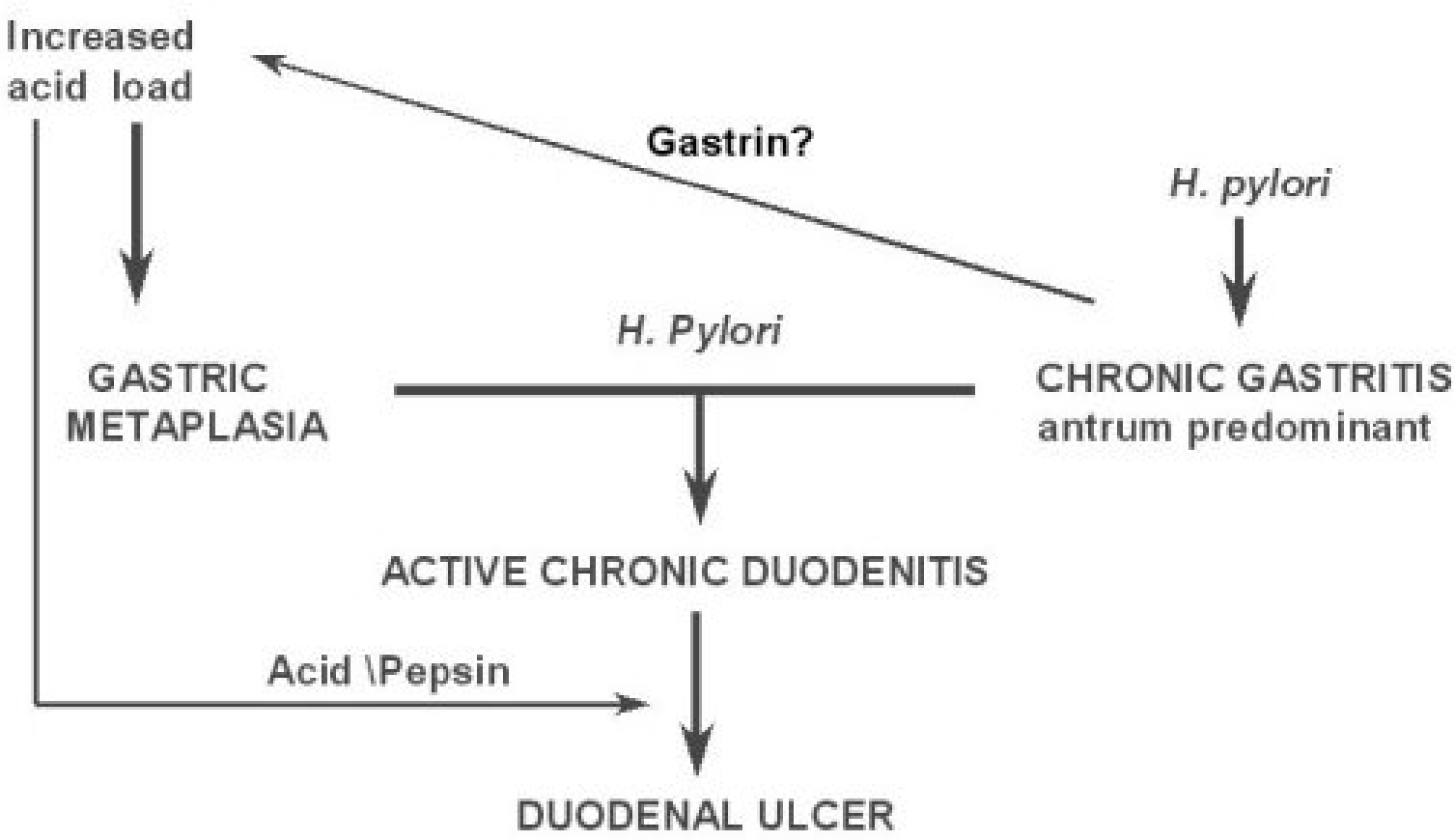
針對不同出血位置加護照護

- 胃12指腸潰瘍出血
  - High dose Proton Pump Inhibitors (PPI)
  - omerprazol(Losec, Prilosec), 40mg bid。
- 靜脈曲張破裂出血
  - 預防肺吸入：
    - endotracheal intubation
    - NG tube intermittent suction to prevent aspiration
    - SB tube intubation
  - 降低門脈壓：
    - Octreotide 50-100ug iv bolus, 25-50ug/h。
  - Endoscopy injection sclerotherapy(EIS)

H. pylori gastritis and duodenal ulcer

- Incidence
  - Major cause of environmental gastritis and peptic ulcer
  - 90%~100% of duodenal ulcer, 70%~90% of gastric ulcer
- Treatment
  - Regimen:
    - Bismuth 8 tablets daily
    - Tetracycline 2 g daily
    - Metronidazole 750 mg daily
  - Duration: 2 weeks
  - May replace with newer antibiotics
- Recurrent of ulcer after healing 10%
- Reinfection rate 1%~2%

H. pylori 引發胃潰瘍或十二指腸潰瘍的可能機轉



* *From J Gastroenterol Hepatol 1991; 6:125-130 (Dixon MF)*

Drugs Used to Treat *H. pylori* Peptic Ulcers

Antibiotics: metronidazole, tetracycline, clarithromycin, amoxicillin

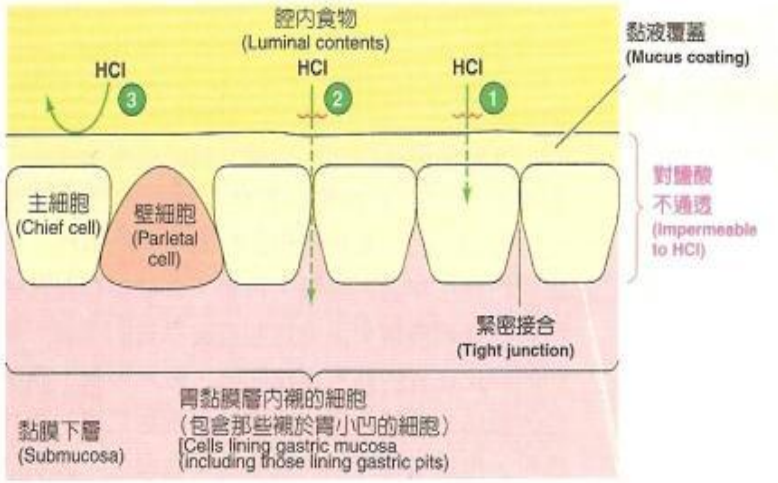
H2 blockers: cimetidine, ranitidine, famotidine, nizatidine

Proton pump inhibitors: omeprazole, lansoprazole, rabeprazole, esomeprazole, pantoprozole

Stomach-lining protector: bismuth subsalicylate

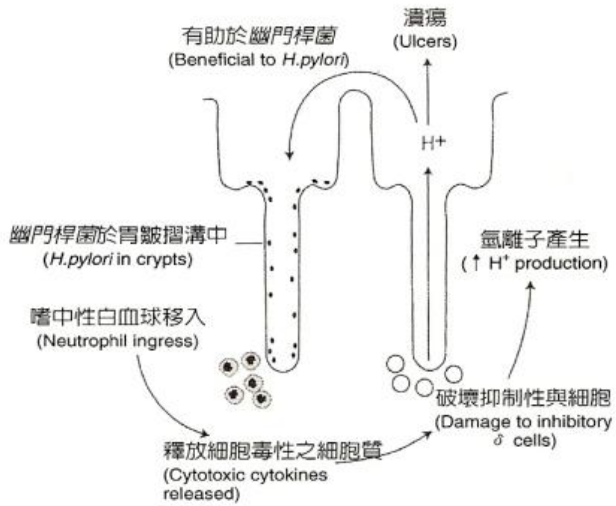
Treatment of peptic ulcer with eradication of *H. pylori*

- **Triple therapy:**
  - **Amoxicillin 500 mg + Metronidazole (Flagyl) 250 mg qid for 2 wks**
  - **One of the followings for 6 wks**
    - **Colloidal bismuth (CBS) 120 mg qid / ac 30' & hs**
    - **Cimetidine 400 mg bid**
    - **Ranitidine 150 mg bid**
    - **Famotidine 20 mg bid**
  - **Amoxicillin 1 gm bid + Clarithromycin 500mg bid + Omeprazloe 20 mg bid for 1 wk. follwed by PPI or H2-antagonist therapy**
- **Dual therapy:**
  - **Amoxicillin 500 mg qid for 2 wks + Omeprazole 20~40 mg q.d. for 4 wks Peptic ulcer**

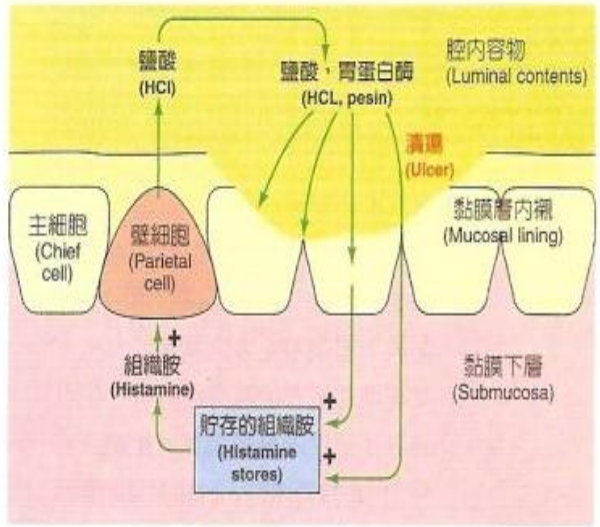


胃黏膜層屏障

胃黏膜層屏障包含以下能讓胃含有酸液，卻不會傷害胃本身的因子：胃黏膜細胞的腔膜對氫離子不通透，所以鹽酸無法穿過細胞 1 ；細胞間是藉由緊密接合的方式連接，所以可預防鹽酸穿透細胞之間 2 ；覆蓋在胃黏膜層的黏液可提供進一步的保護 3 。



幽門桿菌扮演的角色
(ROLE OF H. *PYLORI*)



潰瘍的形成

當酸和胃蛋白酶能夠打破衰弱或擊潰的胃黏膜屏障時，酸能刺激貯存在黏膜下層之組織胺的釋放，組織胺接著再刺激壁細胞分泌更多的酸，而酸再經由破裂的屏障擴散而激起更多的組織胺釋放，惡性循環就這樣繼續下去。當酸和胃蛋白酶持續層損胃黏膜層時，潰瘍形成且漸進性地擴大。

動脈瘤之蜘蛛膜下腔出血
Subarachnoid Hemorrage

Advanced Course

Case Conference

個案狀況

康妮是 42 歲白人女性，在工作時暈倒，之後左側顳葉處嚴重頭痛，因而到急診求診。到急診時，康妮主訴頭痛感覺和之前不同，且當天一開始工作時，便有意識混淆、尿失禁的情形，檢查發現她可以合作行事，但對於環境稍微失去方向感，她的瞳孔兩邊等大且對光均有反應。康妮的先生表示，她頭痛的症狀已經有 3-4 個星期。另外還有噁心、畏光和稍微頸部僵硬的症狀。未含顯影劑的電腦斷層片中顯示在內頸動脈處有蜘蛛膜下腔出血的情形。

康妮在急診初次評估及神經學徵象資料如下：

- BP：152/78mmHg
- BT：37.2℃（99°F）
- HR：102bpm
- Sao2：92% （室內空氣）
- Respirations：30 次/分鐘
- 瞳孔等大且對光有反應
- 嗜睡，但對自己的名字有反應
- GCS：14 分
- 可以按指令動作，四肢活動度一樣

康妮經急診評值後，狀況穩定，入重症照護單位接受照顧。她被安排隔天做動脈血管攝影檢查。並開始以 aminocaproin(Amicar)和 nimodipine(Nimotop)治療。但她仍然抱怨全頭頭痛且頸部僵硬。

康妮在照護單位評估及神經學徵象資料如下：

- BP：142/80mmHg
- BT：37.2℃（99°F）
- HR：84bpm
- Sao2：98%（室內空氣）
- Respirations：24 次/分鐘
- 對人和地點清楚
- 嗜睡，但對自己的名字有反應
- 瞳孔等大且對光有反應
- 可以按指令動作，四肢活動度一樣
- GCS：13 分

康妮在重症加護單位，整晚狀況皆穩定，隔天一早做動脈血管攝影，發現左推側後下小腦動脈瘤。康妮的醫師和家人討論攝影結果並探討如何進一步的治療。康妮做完攝影後神經學狀況依舊穩定，開始做及早手術之準備。

手術

第二天早上十點，康妮進行開顱手術，手術方式是使用夾子將左側下方的小腦動脈瘤夾住。

開刀後狀況

開刀後，康妮的生命徵象立即恢復穩定。她在加護病房內逐漸由全身麻醉中清醒至對名字有反應，且能依指令做動作。康妮在開刀後的數小時中，持續清醒且能辨認出家人。但數小時後康妮突然覺得頭痛、意識混亂且右手整個無力。那時它的生命及神經學徵象如下：

・BP：162/94mmHg	・BT：38.3℃（101°F）
・HR：110bpm	・Respirations：32 次/分鐘
・瞳孔等大且對光有反應	・可以按指令動作，四肢可活動但右手無力抓握
・對人、事、時、地、物不清且有不適當的言語出現	・GCS：11 分

緊急作頭部電腦斷層，片子發現動脈瘤已被夾子綁住、有少許空氣但無明顯積血。經顱都卜勒檢查發現左側中腦動脈的血流速度增加至 120cm/sec，無明顯術後出血情形。再次作動脈血管攝影，結果呈現動脈窄縮。醫師綜合上述結果認為是血管痙攣。她的 Na+＝145mmol/L，K+＝3.9mmol/L，Cl-＝109mmol/L。

再接下來的五天中康妮被打上動脈導管及肺動脈導管（Swan-Ganz）且進行高容液/高灌流的治療方式。康妮的神經學狀況經過此種治療方式後漸穩定。第六至八天，康妮漸漸脫離此種治療方式而神經學狀況仍穩定。隨後被轉至神經科一般病房照護。在一般病房中她的意識狀態警醒且對人、事、時、地、物清楚，但若給予太複雜的指令則易混淆。她可以按指令動作，且四肢皆可活動但右手、右手臂仍稍微無力。她八天後出院且被安置到復健單位持續進行復健治療。

討論題

- Discuss the clinical presentation of a Subarachnoid hemorrhage.
- Describe the incidence, risk factors, mortality, and morbidity associated with acute Subarachnoid hemorrhage.
- Discuss how a Subarachnoid hemorrhage is diagnosed.
- Describe the pathophysiology involved and consequences of blood in the Subarachnoid space.
- Give the rationale for Connie's hyponatremia.
- Describe how a Subarachnoid hemorrhage is classified.

討論題(續)

- What therapeutic modalities should be anticipated for patients after Subarachnoid hemorrhage? Give the rationale for each.
- What parameters require close monitoring for patients after Subarachnoid hemorrhage?
- What are the general complications associated with aminocaproic acid （Amicar）and nimodipine?
- What are the general”aneurysm precautions’ observed for patients with a Subarachnoid hemorrhage who may have a cerebral aneurysm?
- Describe the pathophysiology of cerebral aneurysm.
- Define the different types of aneurysms.

討論題(續)

- Discuss the secondary cerebral injuries associated with a ruptured aneurysm and subsequent Subarachnoid hemorrhage.
- Discuss the advantages and disadvantages of early versus late surgical intervention.
- Discuss why nursing care is of particular importance after a craniotomy.
- Describe the pathophysiology and incidence of rebleeding. When is rebleeding likely to occur?
- Discuss clinical changes that should be anticipated if a patient is experiencing rebleeding.
- Describe how rebleeding would be diagnosed.

討論題(續)

- Discuss the treatment for rebleeding. What is the duration of treatment?
- Describe the pathophysiology and incidence of vasospasm. When is vasospasm likely to occur?
- Discuss the causes of vasospasm.
- Identify how cerebral vasospasm is diagnosed. Include clinical symptoms that should be anticipated.
- Discuss the mortality and morbidity of vasospasm and rebleeding.
- Discuss current treatment for vasospasm. What additional approaches are discussed in the literature?

討論題(續)

- How long should the treatment of vasospasm be continued, and how long will Connie remain at risk for vasospasm or rebleeding?
- Discuss nonsurgical approaches available to patients unable to tolerate traditional surgical treatment.

The clinical presentation of SAH

- sudden, severe, violent headaches
  - unlike any experienced before
  - 40% has headache as warning sign.
- immediate loss of consciousness
- nausea, vomiting
- cranial nerve palsies, and focal neurologic deficits such as weakness of an extremity
- nuchal rigidity or stiff neck,
- photophobia, blurred vision,
- Fever

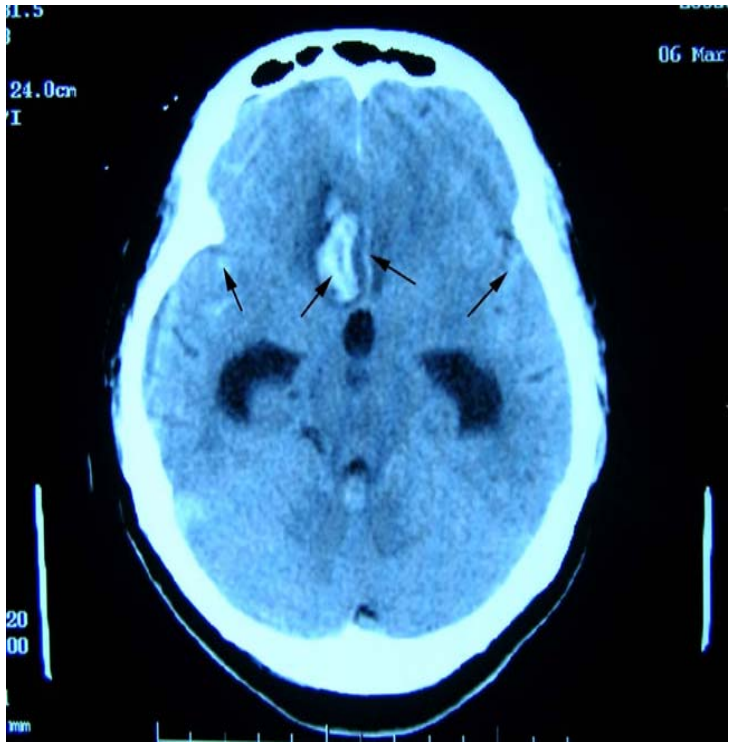
Risk factors of SAH

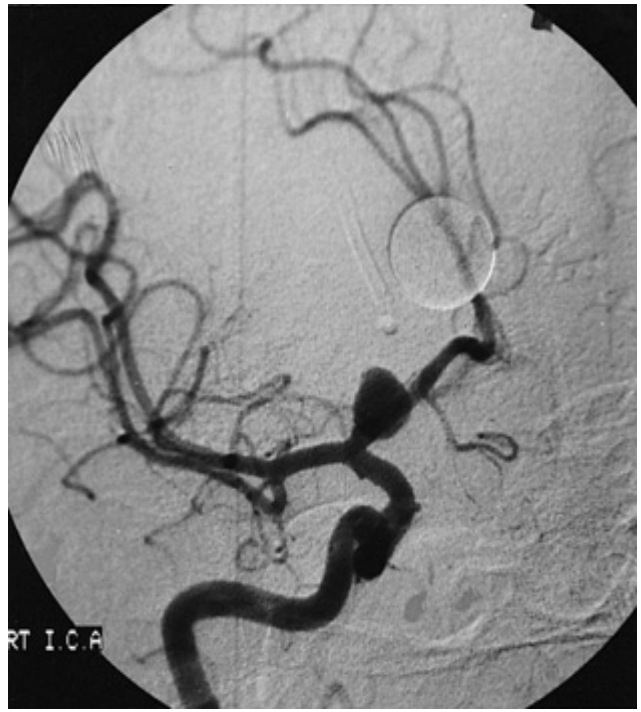
- Age: 35~60 y/o, mean 50
- smoking,
- hypertension,
- alcohol use
- The major causes of death
  - Rebleeding
  - Vasospasm

Diagnosis of SAH

- Lumbar puncture
- CT scan
  - within 48 hours of the bleeding
  - can demonstrate 75% to 85% of the cases.
- Magnetic resonance imaging
- A four-vessel cerebral angiogram
  - Mainstay exam for SAH
  - provide a definitive diagnosis of aneurysm or arteriovenous malformation.
- Cerebral angiography
  - visualizes both intracranial and extracranial vessels.

CT and angiography





Pathophysiology of SAH

- Rupture of aneurysm
  - usually occurs around the circle of Willis at a point of bifurcation of the arterial vessels
- Blood irritates brain tissues, causes inflammatory response and cerebral edema.
  - rapid rise in intracranial pressure
  - reduction of cerebral blood flow
- Hypertensive
  - release of catecholamines
- Neurological injury
  - Blood seeping into adjacent tissue

Rationale for Connie's hyponatremia

- The most common electrolyte imbalance found in patients with SAH (30%)
- probably caused by cerebral salt wasting or the syndrome of inappropriate secretion of antidiuretic hormone (ADH)

Classification of SAH

- The Hunt and Hess classification system
- grade the severity of a subarachnoid bleeding episode in terms of clinical presentation and significance.
  - grade I
    - asymptomatic or minimal headache
  - grade V
    - deep coma, extension abnormal, and moribund appearance

WFNS classification

WFNs Grade	GCS Score	Motor deficit
I	GCS 15	Absent
II	GCS 13-14	Absent
III	GCS 13-14	Present
IV	GCS 7-12	Present or absent
V	GCS 6-3	Present or absent

World Federation of Neurosurgical Societies

Major drug therapy and rationale

- Antifibrinolylic therapv
  - used to prevent clot lysis at the point of rupture to reduce the risk of rebleeding
  - Used during the first 2 weeks and discontinued upon ligation of the aneurysm.
  - Agents such as aminocaproic acid or tranexamic acid
  - may increase hydrocephalus and vasospasm
- Calcium channel blocker
  - to reduce or minimize vasospasm and subsequent secondary ischemia.
  - are associated with hypotension, which can induce cerebral ischemia in the patient after SAH.
  - significantly limit the effectiveness of vasopressor drugs used to maintain systemic blood pressure

Nimodipine

- Currently the drug of choice in SAH
- More potent than nifedipine (Procardia), is lipid soluble, readily crosses the blood-brain barrier, and perhaps has fewer systemic effects from hypotension
- Nimodipine promotes collateral circulation, reduces platelet aggregation, and blocks calcium influx into single nerve cells, thus promoting an antispasmodic effect.
- Nimodipine also enhances the cardiac function by reducing afterload. Treatment is continued for 21 days.
- The typical dosage is 60 mg every 4 hours orally.

Other medications and rational

- Anticonvulsants: to prevent seizures
- Steroids: used if the patient shows signs of IICP, focal neurologic deficits, or cerebral edema.
- Analgesics
  - To control the headaches and pain associated with meningeal irritation.
- Mild sedative: used if the patient is agitated and at risk for IICP
- Stool softener: Valsalva's maneuver may trigger IICP

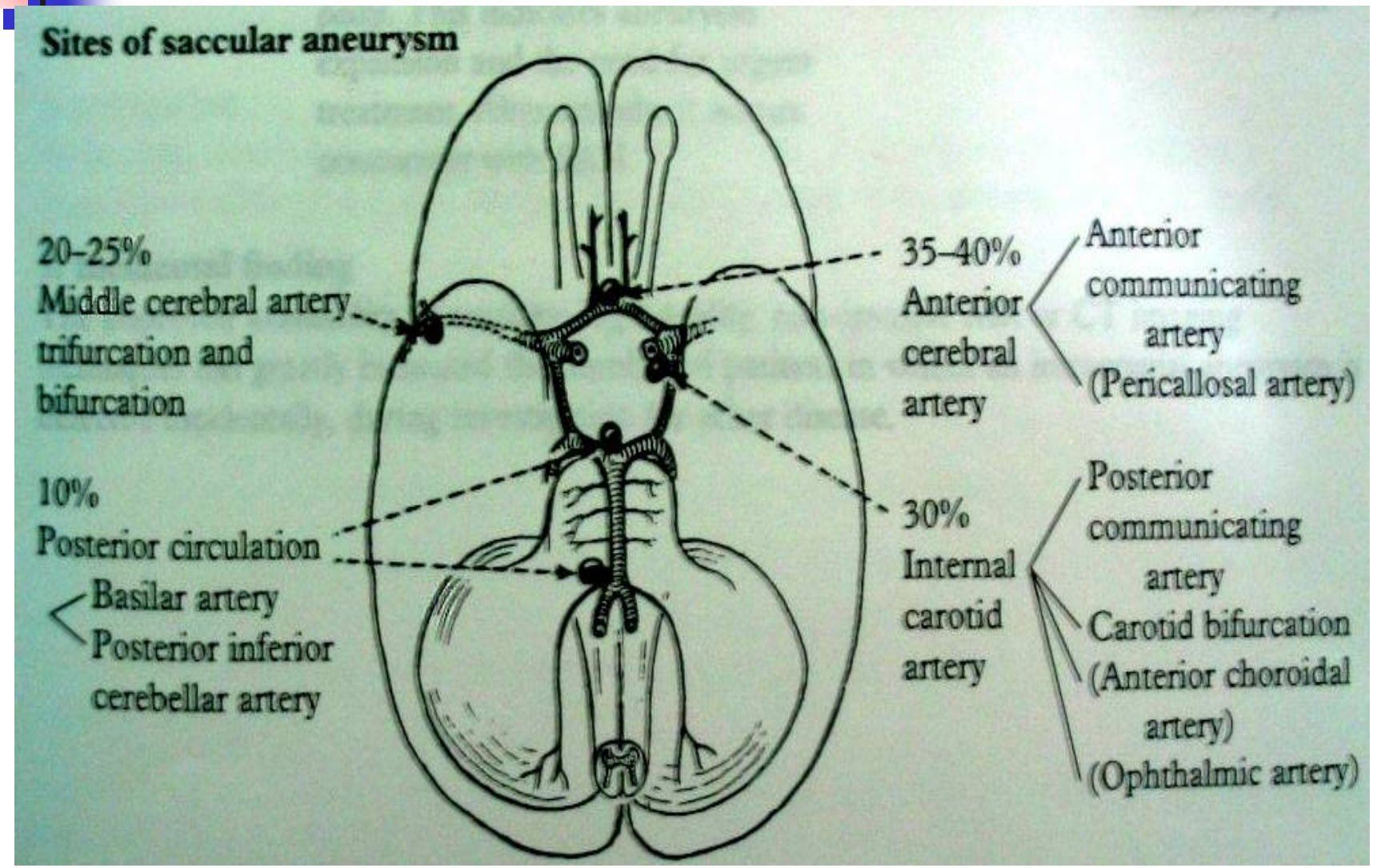
Aneurysm precautions for SAH

- Place the patient in a quiet room
- Maintain bed rest, possibly with bathroom privileges.
- Control the room lighting to prevent strong, direct light.
- Maintain good oxygen and carbon dioxide exchange; supplemental oxygen is usually ordered.
- Maintain head of bed elevation, generally 20 to 30 degrees to augment cerebral venous drainage.
- Avoid overstimulating the patient
  - avoid straining for bowel movements
  - Restrict visitors and discuss with visitors the importance of a calming, supportive environment
  - Schedule nursing care and diagnostic tests.
  - Allow reading and TV watching if these activities do not overstimulate the patient.

Pathophysiology of cerebral aneurysm

- saccular outpouchings of the walls of a cerebral artery.
- Causes unclear
  - Congenital theory
  - Degenerative theory
  - Physical activities or strain

Common site of aneurysm



Secondary cerebral injuries of SAH

- Rebleeding
- Hydrocephalus and IICP
  - Bleeding and clot interfere with the flow and reabsorption of CSF via arachnoid villi
- Seizures
  - Initial seizures occur at the time of the rupture.
  - Early seizures occur within 2 weeks
  - Late seizures usually occur more than 2 weeks after the initial hemorrhage.
  - often followed by rebleeding
  - often phenytom (Dilantin) is the drug of choice
- Vasospasm
- Hypothalamic dysfunction
  - SIADH
  - Water retained, hyponatremia, hypervolemia

Early vs. Late surgery

- Early surgery
  - Prevention of rebleeding and allows for the concomitant removal of intraparenchymal hematoma
  - Enable to aggressive treatment with hypervolemia and hypertension for vasospasm as necessary
  - Technical difficult in doing surgery on an edematous brain
- Late surgery
  - Delay 7~14 days to allow cerebral edema to subside
  - Is better for elderly patients and those with significant comorbidity, for size and location difficult to approach

Key issues for nursing

- Overall and serial evaluation of neurologic status (clot, rebleeding, vasospasm)
- Airway maintanence
- IICP after suction and painful procedures
- Electrolyte and water balance
- Nutrition
- Bed rest
- Pain, esp.headache

Incidence of rebleeding

- Risk for rebleeding
  - Before clip surgery
  - poor premorbid health
  - elevated systolic blood pressures
  - high clinical grade on the Hunt and Hess scale
- Incidence
  - 30% of all patients with aneurysms
  - Peak 24 to 48 hours , duration 28 days,
  - mortality 70%
  - Year rebleeding rate

Incidence of vasospasm

- Correlated with the amount of blood released into the subarachnoid space.
  - high in > 3 by 5 mm by size or > 1 mm by thick in the area of cisterns
- High risk in patients with
  - high clinical grade on the Hunt and Hess scale
  - hyponatremia,
  - hypovolemia,
  - EEG abnormalities,
  - increased cerebral blood flow velocity,
  - decreased regional cerebral blood flow,
  - fibrinogen degradation products greater than 80 μ g/ml in the CSF
- Onset: 3~14 days, peak on day, threat persist for 21 days
- Morbility: 40%, Mortality: 50%

Theories of etiology of vasospasm

- Biochemical process resulting in contraction of arterial muscle cells
- Release of spasmodic substances during the breakdown of erythrocytes and platelets in the subarachnoid blood
- Release of mitogenic substances from platelets, resulting in structural vessel changes
- An inflammatory process resulting in vasculopathy
- oxygen-free radicals

Cerebral vasospasm diagnosis

- S/S
  - lethargy, confusion, restlessness, disorientation, new headaches, hemiparesis, seizures, labile blood pressure, change in speech, elevated fever, or slight leukocytosis,
- Trancranial Doppler
  - vessel narrowing with elevated velocities.
  - Approach from 顳骨(MCA), 眼窩(ACA), and 枕骨大孔(PCA)
  - For mid-verebral artery
    - MV: 50~70 cm/sec, 80~120:mild vasospasm; > 120: severe
    - Pulse Index [(max-min)/mean]: 0.7~0.9, increase in vasospasm

Triple-H therapy for vasospasm

- after the clip ligation of the aneurysm
- to increase cerebral blood flow, cerebral perfusion pressure, and cerebral microcirculation
- Hypertension
  - Dopamine (Intropin) (3 to 10 μ g/kg/min),
  - Dobutamine (Dobutrex) (2.5 to 10 μ g/kg/min),
  - phenylephrine(Neo- Synephrine) (10 to 100 μ g/min)
  - Atropine(prevent bradycardia), Pitressin(prevent diuresis), Digoxin(promote pumping, and prevent heart failure)
- Hypervolemia
  - Palsmanate, Albumine, D5LR, at rate 125~300/hr
- Hemodilution,
  - Maintain Hct around 33%

Guideline during 3-H therapy

- Central venous pressure >10 mm Hg
- PCWP 14 to 20 mm Hg
- Hematocrit 33% to 38%
- Heart rate >70 bpm
- 30% rise in MBP (130 to 150 mm Hg)
- Systolic blood pressure 160 to 200 mm Hg
- Monitor for complications such as
  - pulmonary edema, congestive heart failure, cerebral edema, IICP, myocardial infarction, intracerebral hemorrhage, and peripheral perfusion.

Other treatment methods

- Endovascular Technique（血管內技術）
  - Coil（捲環）栓塞
  - 以4-5個螺旋狀的白金捲環包住動脈瘤基部。此技術需小心血栓的產生。
- 氣球重建術
  - 以一個導管經由頸動脈在放氣球至到動脈瘤處，充氣填塞阻止血液進入，防止動脈瘤破裂。
- 氣球栓塞術
  - 利用電流將鐵絲吸至動脈瘤內，造成動脈瘤栓塞。