

出國報告（出國類別：考察）

衛生福利部臺北醫院
赴蒙古第二中央醫院進行緊急醫療
教學合作交流

蒙古國出國報告書

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出國地區：蒙古國

出國期間：2016年12月12日至12月16日

摘要

衛生福利部臺北醫院臺灣國際醫療衛生人員訓練中心自 2003 年起與蒙古合作進行醫療交流與醫療衛生人員的培訓，並陸續與該國第二中央醫院（Second Central Hospital）、第一婦產科醫院（First Maternity Hospital, Ulaanbaatar）、南戈壁省區域診斷治療中心（The Regional Diagnosis and Treatment Center of South Gobi Province）締結姐妹醫院，此外亦與盧森堡政府發展計畫之蒙古國心臟血管診斷中心（International Project MON/005）簽訂合作備忘錄。

為維繫與蒙古實質之醫療衛生合作交流，鞏固彼此的友好關係，此行由衛生福利部臺北醫院急診室鍾耀文醫師、游秉勳醫師及謝玉蘭護理師，於 2016 年 12 月 12 日至 12 月 16 日期間赴蒙古國辦理緊急醫療教學活動，成功將我國先進之醫療技術及完整醫療教學經驗與蒙古國醫護人員分享，除了有效增進該國醫事人員醫療技術外，更為我國醫療外交拓展一大步。

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壹、目的

衛生福利部臺北醫院臺灣國際醫療衛生人員訓練中心自 2003 年 9 月份培訓第一位來自蒙古國第二中央醫院的內分泌主治醫師起，正式開啟臺蒙兩國醫療交流合作關係。2005 年至蒙古國進行醫療交流合作，與蒙古國第二中央醫院正式締結為姐妹醫院，並於該院舉辦醫護急救訓練班。

延續至今，這段期間內，蒙古國與臺灣之醫療交流不曾中斷過，透過醫療衛生人員培訓及參訪等方式，加深雙邊之合作關係，截至今年為止已成功培訓 315 位蒙古國醫療衛生官員及醫事人員。除了分享臺灣經驗之外，並對醫護人員、醫師提供醫療教學，如高級心肺復甦術（ACLS）訓練等，使其獲得更先進的醫療專業技術與知識，期望能協助蒙古國建立完整的心臟外科手術團隊，提升當地醫療水平。

藉此機會亦邀請過去曾來臺培訓之蒙古學員們相聚一堂，經驗交流並了解學員返國後之發展，學員發表受訓返國後的實際貢獻與成果，學員們均非常感謝臺灣提供培訓機會，增加專業知識及眼界。並勉勵學員們努力貢獻所學，一同為蒙古國醫療衛生盡最大心力。

一、緊急醫療教學團成員

No.	姓名	性別	職稱/服務單位
1	鍾耀文 CHUNG,YAO-WEN	男	衛生福利部臺北醫院急診醫學科醫師 Emergency Doctor of Taipei Hospital, Ministry of Health and Welfare
2	游秉勳 YU,PING-HSUN	男	衛生福利部臺北醫院急診醫學科醫師 Emergency Doctor of Taipei Hospital, Ministry of Health and Welfare
3	謝玉蘭 HSIEH,YU-LAN	女	衛生福利部臺北醫院急診護理師 Nurse of Taipei Hospital, Ministry of Health and Welfare

二、參訪教學行程

天數	日期	行程內容	地點
1	12月12日 星期一	臺北-仁川-烏蘭巴托	烏蘭巴托
2	12月13日 星期二	參訪蒙古第二中央醫院(Second General Hospital)	烏蘭巴托
3	12月14日 星期三	與蒙古第二中央醫院合辦 「醫護人員急救訓練班(ACLS) 」 Hypertensive Emergency	烏蘭巴托
4	12月15日 星期四	與蒙古第二中央醫院合辦 「醫護人員急救訓練班(ACLS) 」 Shock management in emergency care department	烏蘭巴托
5	12月16日 星期五	烏蘭巴托-香港-臺北	

貳、過程

一、參訪蒙古國第二中央醫院 (Second Central Hospital, Mongolia)

該院原為蒙古國第二聯合醫院 (Second General Hospital, Mongolia) 於 2012 年更名為蒙古國第二中央醫院。該院成立於 1931 年，全院約 215 床、共計 411 位員工，其中 89 位為醫師)。該院通過

ISO9001：2000，且進一步於 2011 年度通過 JCI（Joint Commission International）國際醫療品質認證。除了臺灣以外，亦積極與 WHO、UNFPA、JICA、Swanson's Foundation 等國際組織合作。自 2003 年衛生福利部臺北醫院與該院簽訂姐妹醫院以來，已建立多年之合作關係，截至今年，臺灣國際醫療衛生人員訓練中心已成功培訓來自該院 34 位之醫療衛生及行政人員。

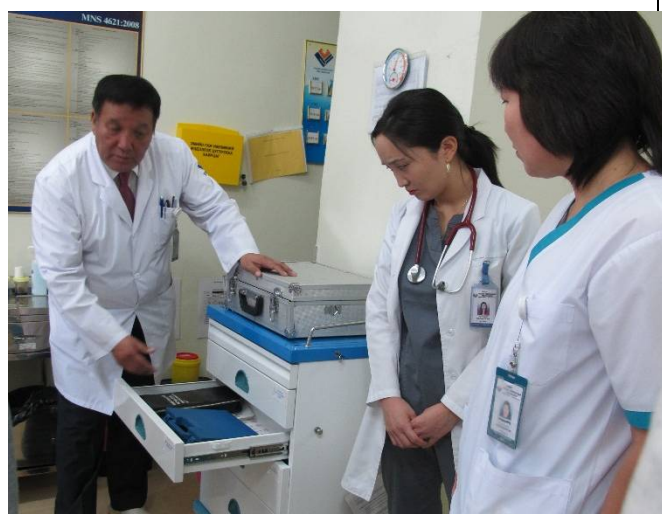
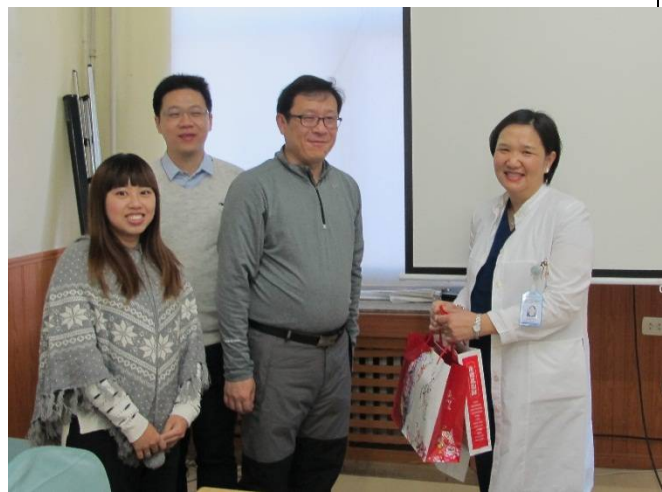
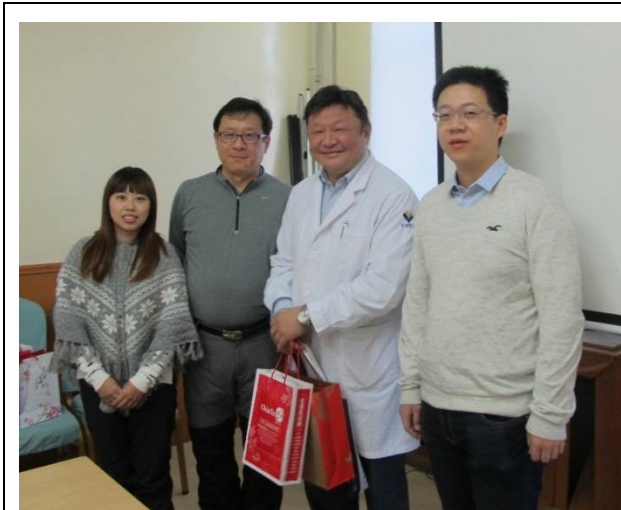
第二中央醫院為當地中大型醫院，與本院及台灣多所醫院有合作關係。該院有加護病房 10 餘床，另有洗腎機約 20 台。該院的加護病房有中央供氧，但並非十分穩定，因此在各個病患的房間內，都有獨立的氧氣筒，以備中央供氧停止時能持續提供病患氧氣。而該院的洗腎機其實不夠當地病患所需，但礙於經費問題，無法大量擴充，該院的洗腎跟台灣不同，台灣通常是早、午、晚三班，但該院只有早午兩班，建議可延長洗腎時間以補足機器無法擴充狀況。

雖然該院的急診室為獨立入口，但沒有方便的通道供救護車直達門口與推床進出，每小時來診病患約 1 人。且沒有獨立的急診專科，急診醫師為各科醫師輪流至急診值班，通常是資淺醫師訓練學習的機會。而急診通往醫院大樓處有電梯直通加護病房門口，但因老舊，常有故障的狀況，電梯故障時則要用人力搬運病患至加護病房（一樓至三樓）。該院的急診有一急救室，設備則相對先進，有急救用藥、氣

管內管、呼吸器、心率監視器等等設備，甚至連超音波都具備，在這樣的硬體設備基礎上，若有足夠的人員訓練，相信能提供病患相當優質的急診服務。但是該院並沒有心導管，因此如果急診遇到心肌梗塞的病患，需要將病患轉診至其他醫院。此外，該院亦無電腦斷層，因此在許多急性病的診斷，例如顱內出血、主動脈剝離等等，恐怕無法即時而有效的診斷。因此也無法進一步的治療。總體來說，該院的急診和內外科，在急重症的處理上，仍有進步的空間。

該院院長非常感謝臺灣衛生福利部及本院長期提供機會，並協助該院行政及醫護人員臨床及專案培訓課程，對於當地醫療衛生技術的進步有功不可沒的貢獻。





二、辦理醫護人員急救教學

此行由本院具有豐富教學經驗的急診醫學科鍾耀文醫師、游秉勳醫師及謝玉蘭護理師於第二中央醫院舉行基本急救（Basic Life Support, BLS）教學課程、高級心肺復甦術（Advance Cardiac Life Support, ACLS）訓練、高血壓急症及敗血症。上午的課程以美國心臟學會（American Heart Association, AHA）之 CPR 與 ECC 準則提要作為主要教材的技術教學課程，除了透過講課、影片播放及親自示範

外，亦提供參與課程的醫護人員實地演練之機會。下午的課程主要介紹急診常見疾病，包括休克與高血壓急症。關於休克，簡介各種休克造成的原因與急診需要做的處理，並重點指出敗血性休克與心因性修課的成因與治療方式。高血壓急症的診斷與處理，則主要指出高血壓急症的成因，與急診需要確診的問題，並且詳述病患後續治療與追蹤之準則。課後亦提供詳細的個案與學員討論，例如主動脈剝離的病患，如何懷疑病患有高血壓急症，並介紹在沒有電腦斷層的狀況下，可以使用哪種輔助檢查與治療。

由於蒙古沒有固定的急診專科醫師，在急診的都是各科的資淺醫師，因此經驗與處理能力上相對較不足。但急診遇到的病患相當多元性，有內科重症、有外傷，或是胃痛腸胃炎的輕症病患，若沒有相當的經驗累積，很難在諸多病患中，抓出有致命性疾病的問題。透過這次的課程，把台灣的急診治療經驗提供對方醫師作為參考，勢必能對該醫院的急診醫療有所助益。

二天課程分別皆有 50 多位踴躍參與，醫護人員們皆認真聽課、發問，表現十分積極，並於課後向本院醫療團隊表達衷心的感谢。





參、心得及建議

蒙古國的平均壽命約 67 歲，相對於台灣的 78~80 歲有約 10 年的落差，反映出兩國在醫療上的不同。該院國際事務中心主任 Dr. Tsendsuren Tumor 特別提到，她的父親因冠心症到台灣接受心導管與支架治療，該治療在蒙古無法實行，亦反映出雙方在醫療技術與設備上的落差。

有關醫院急診醫療的建置方面，建議讓固定的醫師在急診服務，有了固定的人力，才能思考單位需要做什麼改進，若不固定人力，急診的改善較難有連續性的規劃。且可考慮增加超音波來協助診斷，由於超音波是隨時可以使用且不需要裝設大型且昂貴的設備，對該院的急診診斷幫助很大。另外急診是診斷疾病並做初步處理的單位，即便能診斷出急重症，但若沒有能做最終處理的醫師，對病患預後的幫助也是十分有限，必須透過建置完整的醫院醫療體系來改善。

未來，可持續透過可透過我國外交部、駐烏蘭巴托貿易經濟代表處及蒙古駐臺代表處等跨單位之持續聯繫與交流，延續我國與蒙古衛生部的交流合作，以確保交流的穩定與長久，替雙邊的交流創造更多的機會。

更可將曾來臺參訓之醫護人員作為做為種子師資，透過台灣醫療團隊及種子師資教學及示範，與偏遠地區醫護人員最直接交流與指導，

使結訓之蒙古醫護人員可實質回饋當地，並嘉惠一般民眾。

相信經過此次緊急醫療訓練，臺蒙雙邊情誼將更加穩固，在醫衛領域上的交流合作也將愈來愈頻繁。透過提供蒙古國亟需及適切的支援，以強化我國與該國間醫療衛生永續發展關係，進一步拓展臺灣醫療之能見度，呈現我國於醫療衛生的成就，擴展雙方實質與友善的合作關係，促進國際交流合作，延續雙邊醫療衛生永續發展關係。

肆、附件

一、蒙古第二中央醫院邀請函



**SECOND GENERAL
HOSPITAL**

13381 Peace Avenue 49
Bavanzurkh district, Ulaanbaatar, MONGOLIA
Phone/fax: (976) 7015 0200

Date: 2016.06.27
Ref: _____

Dear Director of Taiwan International Healthcare Training Center,

RE: Request for organizing training

It is our pleasure to express our gratitude to your center for making valuable contribution by training our doctors and specialists and showing continued assistance in our work.

In 2014, our hospital carried out restructuring according to the resolution passed by the Minister of Health, and established specialized departments, one of which is the Emergency Department. Within the framework of streamlining the operation of the department and providing quality service to patients, we are in the process of enhancing the skills and qualifications of our doctors and specialists.

In this regard, we request your center's assistance in exploring opportunities in organizing emergency training at our hospital in the third quarter of 2016. State central hospitals, district hospitals, as well as hospitals providing emergency services may also be involved in the training.

Respectfully,

Director Bayasgalan G.

二、教學講義

2 IMPORTANT ADDITIONS

- GRADE: a highly structured and reproducible evidence review system
Grading of Recommendations Assessment, Development, and Evaluation
www.gradeworkinggroup.org
- SEERS: purpose-built AHA Web-based platform
the Systematic Evidence Evaluation and Review system
work together virtually (reviewers from around the world)
ILCOR (visit www.licor.org/seers)



The 2015 AHA Guideline Update for CPR and ECC
Yacwen Chung

UPDATE, NOT A COMPREHENSIVE REVISION

INTRODUCTION

- Summarizes the key issues and changes
- An international evidence evaluation process:
250 evidence reviewers from 39 countries
sufficient new science and controversy to prompt a systemic review
fewer reviews : 2015(166), 2010(274)

CLASS (STRENGTH) OF RECOMMONDATION

- | | |
|------------------------------|------------------|
| • I (STRONG) | Benefit >>> risk |
| • IIa (MODERATE) | Benefit >> risk |
| • IIb (WEAK) | Benefit ≥ risk |
| • III: No benefit (MODERATE) | Benefit = risk |
| • III: Harm (STRONG) | Benefit < risk |

ONLINE

• www.ECCguidelines.heart.org



LEVEL (QUALITY) OF EVIDENCE

- LEVEL A : high quality
- LEVEL B-R: Randomized
- LEVEL B-NR: Nonrandomized
- LEVEL C-LD: Limited Data
- LEVEL C-EO: Expert Opinion

NEW AHA CLASSIFICATION SYSTEM FOR CLASSES OF RECOMMENDATION AND LEVELS OF EVIDENCE

- CLASS (STRENGTH) OF RECOMMONDATION
- LEVEL (QUALITY) OF EVIDENCE

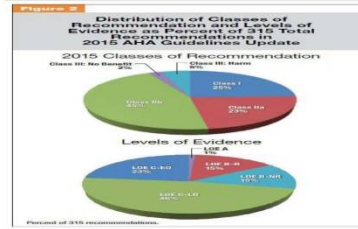
ETHICAL DECISIONS

- The use of extracorporeal CPR (ECPR) for cardiac arrest
- Intra-arrest prognostic factors
- Reviews of evidence about prognostic scores for preterm infants
- Prognostication for children and adults after cardiac arrest
- Function of transplanted organs recovered after cardiac arrest

SYSTEMS OF CARE AND CONTINUOUS QUALITY IMPROVEMENT

- A universal taxonomy of systems of care
- Separation of the AHA adult Chain of Survival into 2 chains: one for in-hospital and one for out-of-hospital systems of care
- Review of best evidence on how these cardiac arrest systems of care reviewed, with a focus on cardiac arrest, ST-segment elevation myocardial infarction (STEMI), and stroke

DISTRIBUTION



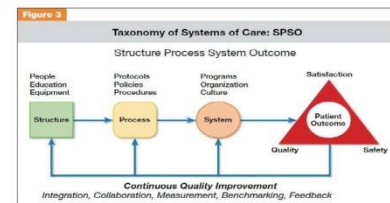
ETHICAL ISSUES

- Healthcare providers (HCPs)
- Provide or withhold
- Whether to start or when to terminate CPR
- In- or out-of-hospital
- Neonatal, pediatrics, adult

USE OF SOCIAL MEDIA TO SUMMON RESCUERS

- Who are willing and able to perform CPR
- Recent study in Sweden: mobile-phone dispatch system
- Significant increase in the rate of bystander-initiated CPR
- Low harm and potential benefit

UNIVERSAL TAXONOMY OF SYSTEMS OF CARE



TEAM RESUSCITATION

- Early warning sign system
- Rapid response teams
- Medical emergency team systems
- Be effective in reducing the incidence of cardiac arrest
- In the general care wards
- Early intervention

THE AHA ADULT CHAIN OF SURVIVAL



HIGH-QUALITY CPR

- Rate :100-120/min (fast)
- Depth : 5(2 inches)-6cm(2.4 inches) (hard)
- Allowing complete chest recoil after each compression
- Minimizing interruptions
- Avoiding excessive ventilation

BEDSIDE

- Team intervention
- Emergency monitoring and resuscitation equipment and drugs
- Trained

HCP BLS

- Match the HCP's clinical setting
- At the same time: checking breathing and pulse
- Team: 1 activate ERS, 2 chest compression, 3 provide ventilation(bag-mask device), 4.set up a defibrillator
- Advanced airway: 1 breath every 6 seconds(10 breaths per minute)

LAY RESCUER CPR

- Out-of-hospital adult chain of survival
- Rescuers activate ERS: mobile telephone, without leaving the victim's side
- Implement PAD programs: communities with people at risk for cardiac arrest(eg, airports, casinos , sports facilities)
- Dispatch-guided CPR
- C-A-B rather than A-B-C; reduce delay to first compression(30:2)
- Bystander-administered naloxone: life-threatening opioid-associated emergencies

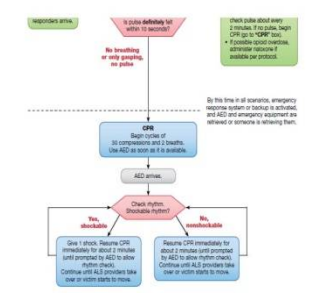
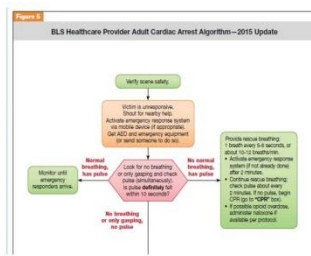


Table 1 BLS Dos and Don'ts of Adult High-Quality CPR

Rescuers Should	Rescuers Should Not
Perform chest compressions at a rate of 100-120/min	Compress at a rate slower than 100/min or faster than 120/min
Compress to a depth of at least 2 inches (5 cm)	Compress to a depth that is too shallow (<5 cm) or greater than 2.4 inches (6 cm)
Allow full chest recoil after each compression	Lean on the chest between compressions
Minimize pauses in compressions	Interrupt compressions for longer than 10 seconds
Provide oxygen (2 breaths after 30 compressions, each breath delivered over 1 second, each lasting about 1 sec)	Provide excessive ventilation (ie, too many breaths or breaths with excessive force)

Table 2 Summary of High-Quality CPR Components for BLS Providers

Component	Adults and Adolescents	Children	Infants
Scene safety	Make sure the environment is safe for rescuer and victim	Make sure the environment is safe for rescuer and victim	Make sure the environment is safe for rescuer and victim
Unresponsive	Unresponsive	Unresponsive	Unresponsive
Normal breathing	Normal breathing	Normal breathing	Normal breathing
Normal breathing and pulse	Normal breathing and pulse	Normal breathing and pulse	Normal breathing and pulse
Normal breathing, no pulse	Normal breathing, no pulse	Normal breathing, no pulse	Normal breathing, no pulse
No normal breathing, but a pulse	No normal breathing, but a pulse	No normal breathing, but a pulse	No normal breathing, but a pulse
No normal breathing, no pulse	No normal breathing, no pulse	No normal breathing, no pulse	No normal breathing, no pulse
Check pulse	Check pulse about every 2 minutes. If possible, high-quality CPR. • If possible, avoid rescue breathing. • Provide a 2-minute rest if you are the sole rescuer.	Check pulse about every 2 minutes. If possible, high-quality CPR. • If possible, avoid rescue breathing. • Provide a 2-minute rest if you are the sole rescuer.	Check pulse about every 2 minutes. If possible, high-quality CPR. • If possible, avoid rescue breathing. • Provide a 2-minute rest if you are the sole rescuer.
Call for help	Call for help	Call for help	Call for help
Begin CPR	Begin CPR (30:2)	Begin CPR (30:2)	Begin CPR (30:2)
Check rhythm	Check rhythm (Shockable rhythm?)	Check rhythm (Shockable rhythm?)	Check rhythm (Shockable rhythm?)
Shockable rhythm	Shock 1 shock. Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.	Shock 1 shock. Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.	Shock 1 shock. Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.
Not shockable rhythm	Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.	Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.	Resume CPR immediately for about 2 minutes until prompted by AED to deliver another rhythm shock. Continue until AED provides final cue or victim starts to move.
Obtain second rhythm	Obtain second rhythm	Obtain second rhythm	Obtain second rhythm
Obtain second shockable rhythm	Obtain second shockable rhythm	Obtain second shockable rhythm	Obtain second shockable rhythm
Obtain second not shockable rhythm	Obtain second not shockable rhythm	Obtain second not shockable rhythm	Obtain second not shockable rhythm

ACUTE CORONARY SYNDROMES

- Prehospital ECG
- Reperfusion: transport to a PCI center.

ADULT ACLS

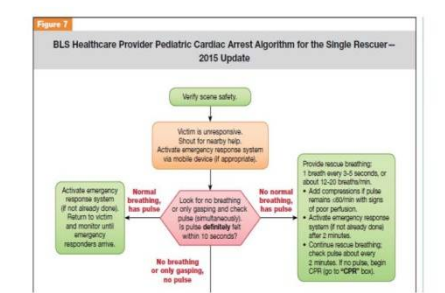
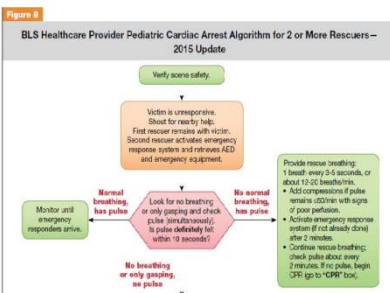
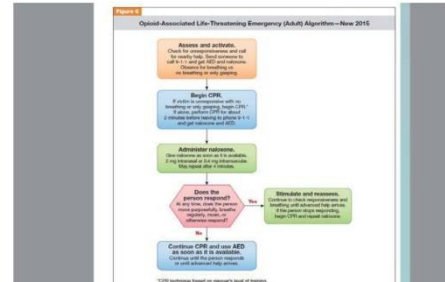
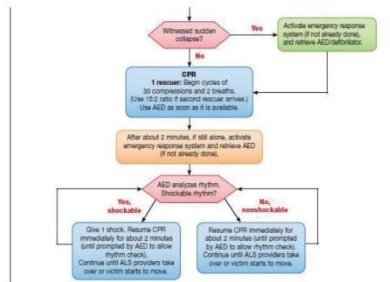
- Vasopressin removed
- Low ET_{CO}₂: CPR > 20 minutes, (not) isolation used for terminating CPR (combination with other factors)
- ECPR: provide time to treat potentially reversible conditions or arrange for cardiac transplantation
- Early provision of epinephrine
- Lidocaine : not routine use, but ROSC from VF/pulseless VT cardiac arrest.
- B-blocker: early after hospitalization from cardiac arrest due to VF/pVT

Box 2 Useful Clinical Findings That Are Associated With Poor Neurologic Outcome*

- Absence of pupillary reflex to light at 72 hours or more after cardiac arrest
 - Presence of status myoclonus (different from isolated myoclonic jerks) during the first 72 hours after cardiac arrest
 - Absence of the N20 somatosensory evoked potential cortical wave 24 to 72 hours after cardiac arrest or after rearming
 - Presence of a marked reduction of the gray-white ratio on brain CT obtained within 2 hours after cardiac arrest
 - Extensive restriction of diffusion on brain MRI at 2 to 6 days after cardiac arrest
 - Persistent absence of EEG reactivity to external stimuli at 72 hours after cardiac arrest
 - Persistent burst suppression or intractable status epilepticus on EEG after rearming
- Absent motor movements, extensor posturing, or myoclonus should not be used alone for predicting outcome.
- *Shock, temperature, metabolic derangement, prior sedatives or neuromuscular blockers, and other clinical factors should be considered carefully because they may affect results or interpretation of some tests.
- Abbreviations: CT, computed tomography; EEG, electroencephalogram; MRI, magnetic resonance imaging.

POST-CARDIAC ARREST CARE

- Emergency coronary angiography: ST elevation, a cardiovascular lesion is suspected (unstable)
- TTM: all comatose adult patients with ROSC after cardiac arrest, targeted temperature management, 32–36 °C, at least 24 hours (old, 32–34 °C, 12–24 hrs)
- Out-of-hospital cooling: not recommended
- Avoid hypotension and correct it immediately (SBP < 90 mm Hg, mean < 65 mm Hg)
- Organ donation: kidney or liver



<p style="text-align: center;">Case 1</p> <ul style="list-style-type: none"> • Past medical history: <ul style="list-style-type: none"> – Hypertension with poor medical adherence – Denied history of diabetes mellitus, coronary disease 	<p style="text-align: center;">Hypertensive emergency</p> <p style="text-align: center;">Yu, Ping-Hsun, MD Emergency department, Taipei hospital</p>
<p style="text-align: center;">Case 1</p> <ul style="list-style-type: none"> • What's your impression (possible diagnosis)? • What's physical examination would you focus on? • What's exam would you like to arrange? <ul style="list-style-type: none"> – Lab – Image – EKG • Should BP be controlled? <ul style="list-style-type: none"> – If so, what agent would you use? 	<p style="text-align: center;">Case 1</p> <ul style="list-style-type: none"> • 62-year-old man • Clinical presentation <ul style="list-style-type: none"> – Sudden onset chest pain with radiation to back – Cold sweats, left upper extremity numbness • Vital signs: <ul style="list-style-type: none"> – Consciousness: clear and oriented, E4V5M6 – BP: 192/122 – HR: 88 – Temp: 37.2 °C

<p style="text-align: center;">Case 2</p> <ul style="list-style-type: none"> • What's your impression (possible diagnosis)? • What's physical examination would you focus on? • What's exam would you like to arrange? <ul style="list-style-type: none"> – Lab – Image – EKG • Should BP be controlled? <ul style="list-style-type: none"> – If so, what agent would you use? 	<p style="text-align: center;">Case 2</p> <ul style="list-style-type: none"> • 52-year-old woman • Clinical presentation <ul style="list-style-type: none"> – Slurred speech, right-sided weakness since wake up today • Vital signs: <ul style="list-style-type: none"> – Consciousness: clear and oriented, E4V5M6 – BP: 240/185 – HR: 55 – Temp: 36.4 °C
<p style="text-align: center;">Case 3</p> <ul style="list-style-type: none"> • 78-year-old man • Clinical presentation <ul style="list-style-type: none"> – Progressive dyspnea for 7~8 hours – Orthopnea • Vital signs: <ul style="list-style-type: none"> – Consciousness: clear and oriented, E4V5M6 – BP: 210/150 – HR: 82 – Temp: 36.6 °C 	<p style="text-align: center;">Case 2</p> <ul style="list-style-type: none"> • Past medical history: <ul style="list-style-type: none"> – Hypertension and diabetes mellitus with poor medical adhesion

<p style="text-align: center;">Definition</p> <ul style="list-style-type: none"> • Hypertension • Hypertensive urgency • Hypertensive emergency 	<p style="text-align: center;">Case 3</p> <ul style="list-style-type: none"> • Past medical history: <ul style="list-style-type: none"> – Hypertension with poor medical adherence – Rheumatic heart disease with severe mitral regurgitation (MR)
<p style="text-align: center;">Hypertension</p> <ul style="list-style-type: none"> • JNC 	<p style="text-align: center;">Case 3</p> <ul style="list-style-type: none"> • What's your impression (possible diagnosis)? • What's physical examination would you focus on? • What's exam would you like to arrange? <ul style="list-style-type: none"> – Lab – Image – EKG • Should BP be controlled? <ul style="list-style-type: none"> – If so, what agent would you use?

<p style="text-align: center;">Epidemiology</p> <ul style="list-style-type: none"> • Acute hypertensive emergency <ul style="list-style-type: none"> – Mostly in patients with known hypertension without adherence to regular antihypertensive regimen • 1% ~2% patients with hypertension will develop hypertensive emergency • 0.5% ~ 3% of total ED (emergency department) visit due to hypertensive emergency 	<p style="text-align: center;">Hypertensive emergency</p> <ul style="list-style-type: none"> • Acute elevation of BP AND end organ damage • BP> 180/120 mmHg • End organ damage, acute one <ul style="list-style-type: none"> – Heart – Aorta – Kidney – Brain – Eyes
<p style="text-align: center;">Pathophysiology</p> <ul style="list-style-type: none"> • Systemic vasoconstrictor → elevation of systemic vascular resistance → endothelial injury → activation of coagulation cascade • Clinical presentation <ul style="list-style-type: none"> – Hematuria (kidney) – Arterial hemorrhage or exudate (eye) 	<p style="text-align: center;">Hypertensive urgency</p> <ul style="list-style-type: none"> • Acute elevation of BP without end organ damage • BP> 180/120 mmHg • Although clinical benefit of treatment is unclear, BP over 180/120 is often cited as an indication for treatment

<p style="text-align: center;">Chest pain and severe hypertension</p> <ul style="list-style-type: none"> • Aortic dissection should be suspected <ul style="list-style-type: none"> – sudden onset of unexplained chest pain that radiates to the back – sudden onset of pain associated with any of the associated signs and symptoms <ul style="list-style-type: none"> • Neurological signs • Pulse deficit 	<p style="text-align: center;">Clinical feature</p> <ul style="list-style-type: none"> • Chest pain and severe hypertension • Acute neurological symptoms and severe hypertension • Peripheral edema and severe hypertension • Sympathetic crisis and severe hypertension • Asymptomatic and benign severe hypertension
<p style="text-align: center;">Acute neurological symptoms and severe hypertension</p> <ul style="list-style-type: none"> • Hypertensive encephalopathy <ul style="list-style-type: none"> – altered mental status – headache, vomiting, seizures, or visual deficit • Stroke <ul style="list-style-type: none"> – Ischemic or hemorrhagic – Focal neurological signs 	<p style="text-align: center;">Chest pain and severe hypertension</p> <ul style="list-style-type: none"> • Aortic dissection <ul style="list-style-type: none"> – abrupt, severe onset of pain (90% of cases), usually in the chest (78% of cases) <ul style="list-style-type: none"> • tearing or ripping, and radiating to the interscapular region – diastolic murmur (28%) – neurologic deficits (17%) – Chest radiograph abnormality (90%) <ul style="list-style-type: none"> • radiographic signs are multiple and not specific for aortic dissection – ECG changes 25% <ul style="list-style-type: none"> • 4% percent have ST elevation in two or more contiguous leads • Acute coronary syndrome

<p style="text-align: center;">Asymptomatic and benign severe hypertension</p> <ul style="list-style-type: none"> • headache, visual changes, chest pain, dyspnea, and dizziness • Poor correlation between severity of hypertension and symptoms 	<p style="text-align: center;">Peripheral edema and severe hypertension</p> <ul style="list-style-type: none"> • Acute renal failure <ul style="list-style-type: none"> – Edema, oliguria, poor appetite • Preeclampsia <ul style="list-style-type: none"> – Pregnancy, edema and proteinuria – May have elevated liver enzyme level, low platelet and hemolysis
<p style="text-align: center;">Diagnosis</p> <ul style="list-style-type: none"> • Aortic dissection <ul style="list-style-type: none"> – CT or transesophageal echocardiography • Acute coronary syndrome <ul style="list-style-type: none"> – Abnormal EKG or/and cardiac enzyme elevation • Acute pulmonary edema <ul style="list-style-type: none"> – Chest radiography • Severe preeclampsia <ul style="list-style-type: none"> – Pregnancy, proteinuria, elevated live enzyme level, low platelet, seizure 	<p style="text-align: center;">Sympathetic crisis and severe hypertension</p> <ul style="list-style-type: none"> • Pheochromocytoma <ul style="list-style-type: none"> – Alternate period of symptoms – Headache, tachycardia and flush skin • Illicit drug use <ul style="list-style-type: none"> – Amphetamine, cocaine – tachycardia, diaphoresis, and hypertension – mental status changes

Diagnosis

- ED-identified hypertension
 - No large study about the validity of ED BP screen
 - Several small studies found the association between ED blood pressure elevation and chronic hypertension
 1. Pitts SR, Adams RP: Emergency department hypertension and regression to the mean. *Ann Emerg Med* 31: 214, 1998
 2. Chiang WK, Jamshehi B: Asymptomatic hypertension in the ED. *Am J Emerg Med* 16: 701, 1998
 3. Mamon J, Green L, Levine DM, et al: Using the emergency department as a screening site for high blood pressure. A method for improving hypertension detection and appropriate referral.
 4. Dieterle T, Schuurmans MM, Strobel W, et al: Moderated-to-severe blood pressure elevation at ED entry: hypertension or normotension. *Am J Emerg Med* 23: 474, 2005

Diagnosis

- Acute renal failure
 - Serum creatinine level
- Hypertensive encephalopathy
 - clinical diagnosis, including altered mental status associated with elevated blood pressure
 - Exclusion of alternative diagnosis (brain CT)
- SAH (subarachnoid hemorrhage), ICH (intracranial hemorrhage)
 - Brain CT (computed tomography)

Treatment of asymptomatic hypertension and hypertensive urgency

- Association between acute BP control and long-term outcome was uncertain
- may improve some minor, not life-threatening conditions, as dizziness, headache
- Nifedipine
- Captopril

Diagnosis

- Ischemic stroke
 - Clinical diagnosis, new onset neurological sign, exclude other causes
- Sympathetic crisis
 - Clinical diagnosis
 - Drug screen for illicit drug user
 - Pheochromocytoma: 24-h urine test for catecholamines and metanephrine

Treatment - aortic dissection

Agents	Relative effect based on BP and PVL target (SBP < 100-120 mm Hg, PVL < 10 mm) or SBP < 160 mm Hg, PVL < 10 mm	Relative effect based on SBP and PVL target (SBP < 100-120 mm Hg, PVL < 10 mm) or SBP < 160 mm Hg, PVL < 10 mm	Relative effect based on SBP and PVL target (SBP < 100-120 mm Hg, PVL < 10 mm) or SBP < 160 mm Hg, PVL < 10 mm	Relative effect based on SBP and PVL target (SBP < 100-120 mm Hg, PVL < 10 mm) or SBP < 160 mm Hg, PVL < 10 mm
Hydralazine	Hydralazine 25-50 mg IV q4-6h	Hydralazine 25-50 mg IV q4-6h	Hydralazine 25-50 mg IV q4-6h	Hydralazine 25-50 mg IV q4-6h
Labetalol	Labetalol 20-30 mg IV q4-6h	Labetalol 20-30 mg IV q4-6h	Labetalol 20-30 mg IV q4-6h	Labetalol 20-30 mg IV q4-6h
Esmolol	Esmolol 50-100 mcg/kg/min IV infusion	Esmolol 50-100 mcg/kg/min IV infusion	Esmolol 50-100 mcg/kg/min IV infusion	Esmolol 50-100 mcg/kg/min IV infusion
Verapamil	Verapamil 2-4 mg IV q4-6h	Verapamil 2-4 mg IV q4-6h	Verapamil 2-4 mg IV q4-6h	Verapamil 2-4 mg IV q4-6h
Nitroglycerin	Nitroglycerin 0.25-1 mg IV q4-6h	Nitroglycerin 0.25-1 mg IV q4-6h	Nitroglycerin 0.25-1 mg IV q4-6h	Nitroglycerin 0.25-1 mg IV q4-6h

Treatment of asymptomatic hypertension and hypertensive urgency

Medication	Dose	Onset of Action	Duration	Contraindications	Adverse Effects
Nitroglycerin	Hydralazine 25-50 mg IV q4-6h	0.5-1 h	8-12 hrs	Aortic dissection	Headache
Labetalol	1-2 Adrenergic blocker	30-60 min	8-12 h	Aortic, aortic dissection, pulmonary edema, hypotension	Bradycardia, hypotension
Clonidine	Central alpha agonist	30-60 min	8-12 h	CHF, severe or third degree heart block	Drowsiness, sedation, hypotension, dry mouth
Captopril	Angiotensin-converting enzyme inhibitor	30-60 min	4-6 h	Renal artery stenosis, pregnancy	Acute renal failure, angioedema
Nifedipine (immediate release)	Calcium channel blocker	15-30 min	8-12 h	Angina, acute hypotension	Hypotension, reflex tachycardia, peripheral edema, dizziness, heart block, CHF
Levamisole	Angiotensin II antagonist	30 min	12-24 h	Renal and fetal distress of pregnancy	Angina, muscle cramps

Treatment – hypertensive pulmonary edema

- IV NTG (nitroglycerine) and diuretics
- consider beta-blocker if pulmonary edema resulted from
 - Af (atrial fibrillation) with RVR (rapid ventricular response)
 - ACS (acute coronary syndrome)

Treatment - aortic dissection

- Blood pressure
 - SBP: 100~120, <140 at least
- HR: <60 /min
- **ALWAYS** use beta-blocker before vasodilator
 - Shearing force will increase if tachycardia
 - Reflex tachycardia while using vasodilator alone
 - Labetalol
 - NTG (nitroglycerin)

Treatment – acute sympathetic crisis

Acute sympathetic crisis (acute hypertension, MCH toxicity)	Routine essential antihypertensive and symptomatic relief		Labetalol is contraindicated if given with other drugs with a beta-blocker
		Nifedipine ²⁰ IV bolus	Rebound/bradycardia may be severe, observe for respiratory depression
		Nitroglycerin 0.5-1mg IV continuous infusion ²¹	
		Phenylephrine ²² IV or IM	
		Hydroxyzine IV or oral Morphine ²³	

Treatment – hypertensive pulmonary edema

Acute hypertensive pulmonary edema	Reduce BP by 10%-20%, diuretic through mechanical, symptomatic relief			
		Nifedipine 0.5-1mg IV continuous infusion ²⁰		IV diuretic (furosemide) essential if low sodium, high for those with chronic renal and low BP
		Furosemide IV ²⁴	ACE inhibitors, non-vasodilator function	Avoid hypotension
		Nitroglycerin IV continuous infusion		Use with caution, severe bradycardia, hypotension a regular through effect
		Hydroxyzine IV or oral Morphine ²³		Caution and titration needed in patients with reduced renal function or chronic renal failure
		Diuretics IV ²⁴		Monitor sodium, potassium and urea

Treatment – eclampsia and preeclampsia

- Labetalol, nifedipine
- ACEI is contraindicated
 - Teratogen
- Hydralazine
 - Not recommended now
 - Unpredicted effect

Treatment – acute sympathetic crisis

- Reduction of sympathetic drive
- Benzodiazepine (BZD): lorazepam, midazolam
 - Monitor respiration while BZD use
- Unopposed beta-blocker can induce alpha-storm
- NTG, calcium channel blocker (CCB) if still severe hypertension under BZD

Treatment – intracranial hemorrhage

- If SBP > 200 mg, consider IV infusion medication for BP control
- Keep SBP < 160 mmHg
 - If ICP (increased intra-cranial pressure) sign presents → consult neurosurgeon earlier and ICP monitor consider
 - No increased morbidity or mortality while keep SBP: 120~160 mmHg
- Nicardipine, Labetalol

Treatment – hypertensive encephalopathy

- Lowering MAP (mean arterial pressure) 20~25% in the first hour
 - Too aggressive BP lowering may lead to ischemic infarction

Treatment – intracranial hemorrhage

Intracranial hemorrhage	SBP > 180 or MAP > 130 mmHg, consider aggressive management, IV labetalol ²⁵		Check for SBP < 160 mmHg and not associated with increased morbidity ²⁶
	SBP > 180 or MAP > 130 mmHg and possible expansion of ICH, use labetalol IV for longer while maintaining CVP > 60 mmHg		Early hemorrhage growth often occurs in the 1st 6 hours after rupture and acute BP rise, requires BP control (SBP < 160, or 140 mmHg otherwise, depending on age, comorbidity, and severity) ^{26,27}
	SBP > 180 or MAP > 130 mmHg and no evidence of ICH, give SBP < 170 mmHg (150 mmHg)	Labetalol ²⁵ IV bolus or continuous infusion	
		Nifedipine ²⁰ IV or oral	
		Furosemide ²⁴ IV bolus, then continuous infusion	

Treatment – hypertensive encephalopathy

Hypertensive encephalopathy	Control SBP 20% in the first hour of presentation ²⁸ , consider aggressive lowering may lead to infarction/bleeds			Adjustability of several particular drugs to individual patients, an exact target BP should be sought, do not give nifedipine ²⁰ as a long-acting calcium channel blocker
		Nifedipine ²⁰ IV continuous infusion		
		Labetalol ²⁵ IV continuous infusion		Avoid hypotension while brain edema
		Furosemide ²⁴ IV continuous infusion		
		Diuretics IV continuous infusion ²⁴		

shock

Yu, Ping-Hsun, MD
Emergency department, Taipei hospital

Pathophysiology

- Circulatory insufficiency → imbalance between tissue oxygen demand and supply
- Systemic oxygen delivery
 - Cardiac output
 - arterial oxygen content

Epidemiology

- Incidence rate: 135~217/100,000
- High mortality rate, 1-month mortality
 - 30~45% in patient with septic shock
 - 60~90% in patients with cardiogenic shock

Shen HN, et. Chest 2010; 138:298-304

Pathophysiology

- Oxygen content in blood
- $CaO_2 = 0.0031 * PaO_2 + 1.38 * Hb * SaO_2$
 - CaO_2 : amount of oxygen in 100 mL of blood
 - PaO_2 : arterial oxygen pressure
 - SaO_2 : arterial oxygen saturation
- SaO_2 : pulse oximetry
- PaO_2 : arterial blood gas

Pulse oximetry



oxygen saturation

Pathophysiology

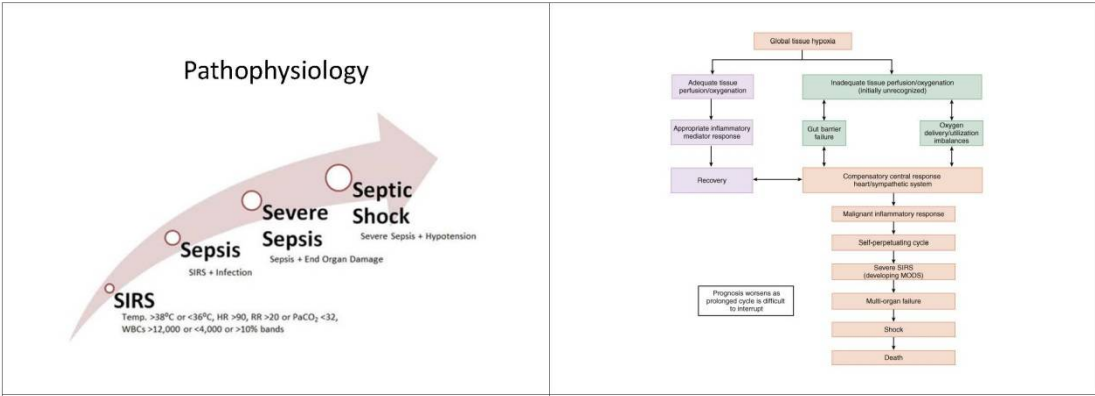
- Compensation mechanisms → maintain blood flow in vital organs, brain and heart
 - Vasoconstriction
 - Increased heart rate
 - Release vasoactive hormone
 - Epinephrine, norepinephrine
 - Activation of renin-angiotensin system
 - Maintain intravascular volume

Pathophysiology

- Inadequate oxygen supply → compensation
 - Increased cardiac output
 - Increased extraction fraction of oxygen in hemoglobin
 - Decreased venous oxygen saturation
- Compensation failure → anaerobic respiration
 - Lactate formation (lactic acidosis)

Pathophysiology

- Cellular response
 - influx of sodium, efflux of potassium, and reduction in membrane resting potential.
 - lysosomal enzymes are released into the cells with subsequent hydrolysis of membranes
 - loss of cellular integrity and the breakdown in cellular homeostasis result in cellular death
- Clinical finding
 - hemoconcentration
 - hyperkalemia,
 - hyponatremia
 - prerenal azotemia
 - hyper- or hypoglycemia
 - lactic acidosis



Clinical feature

- History:
 - Often, the cause is apparent
 - Hemorrhagic: GI bleeding, severe diarrhea
 - Cardiogenic: acute myocardial infarction (AMI)
 - Septic: high fever, large wound with infection
 - Patients with neurological dysfunction are more susceptible to hypovolemia

Clinical feature

- Physical examination
 - Usually, systolic BP < 90 mmHg
 - Insensitivity of BP to detect tissue hypo-perfusion
 - Normal BP with shock
 - Hypotension without shock
 - Shock index
 - Heart rate/systolic blood pressure
 - Normal range: 0.5~0.9
 - > 0.9 indicate impaired left ventricular (LV) function and high mortality

Clinical feature

- Temperature:
 - hypothermia or hyperthermia
- Heart rate:
 - usually elevated
- Blood pressure:
 - may increased initially then fall after shock progress
- Central nervous system (CNS)
 - Acute delirium, restlessness, disorientation, confusion, and coma

Clinical feature

- Cardiovascular
 - Tachycardia, neck vein distended or flatten, pulmonary edema
- Skin
 - Pale, pallor, cyanosis, sweating
- Metabolic
 - Respiratory alkalosis initially, then metabolic acidosis while shock progress

Shock category

Type	Comment	Examples
Cardiogenic	Inadequate heart pump function	Heart failure due to acute myocardial infarction (AMI)
Hypovolemic	Inadequate circulation volume	Massive gastrointestinal (GI) bleeding
Obstructive	Extra-cardiac obstruction of blood flow	Pulmonary embolism, tension pneumothorax
Distributive	Cellular respiration impairment due to metabolic derangements	Septic shock, cyanide intoxication

Laboratory examination

- Basic exam
 - CBC, electrolytes, glucose, serum creatinine, thrombin time (PT), partial thrombin time (PTT), chest radiography, EKG
- Physiological assessment
 - serum lactate, blood gas, fibrinogen, D-dimer
- Non-invasive hemodynamic monitor
 - Cardiac echo, end-tidal CO₂

<p style="text-align: center;">Chest pain and severe hypertension</p> <ul style="list-style-type: none"> • Aortic dissection should be suspected <ul style="list-style-type: none"> – sudden onset of unexplained chest pain that radiates to the back – sudden onset of pain associated with any of the associated signs and symptoms <ul style="list-style-type: none"> • Neurological signs • Pulse deficit 	<p style="text-align: center;">Clinical feature</p> <ul style="list-style-type: none"> • Chest pain and severe hypertension • Acute neurological symptoms and severe hypertension • Peripheral edema and severe hypertension • Sympathetic crisis and severe hypertension • Asymptomatic and benign severe hypertension
<p style="text-align: center;">Acute neurological symptoms and severe hypertension</p> <ul style="list-style-type: none"> • Hypertensive encephalopathy <ul style="list-style-type: none"> – altered mental status – headache, vomiting, seizures, or visual deficit • Stroke <ul style="list-style-type: none"> – Ischemic or hemorrhagic – Focal neurological signs 	<p style="text-align: center;">Chest pain and severe hypertension</p> <ul style="list-style-type: none"> • Aortic dissection <ul style="list-style-type: none"> – abrupt, severe onset of pain (90% of cases), usually in the chest (78% of cases) <ul style="list-style-type: none"> • tearing or ripping, and radiating to the interscapular region – diastolic murmur (28%) – neurologic deficits (17%) – Chest radiograph abnormality (90%) <ul style="list-style-type: none"> • radiographic signs are multiple and not specific for aortic dissection – ECG changes 25% <ul style="list-style-type: none"> • 4% percent have ST elevation in two or more contiguous leads • Acute coronary syndrome

<p style="text-align: center;">Treatment</p> <ul style="list-style-type: none"> • Controlling work of breathing <ul style="list-style-type: none"> – Respiratory muscle significantly increased oxygen consumption while shock – Mechanical ventilation and sedation decreased respiration work load and improve survival – Goal <ul style="list-style-type: none"> • PaCO₂: 35~40 mmHg • SpO₂: >93% 	<p style="text-align: center;">Treatment</p> <ul style="list-style-type: none"> • Optimize circulation <ul style="list-style-type: none"> – First resuscitation with isotonic crystalloid – 500 mL ~ 1000 mL bolus, then reassessment – Vasopressors, used initially to prevent adverse consequence of systemic hypotension <ul style="list-style-type: none"> • Epinephrine • Dopamine
<p style="text-align: center;">Treatment</p> <ul style="list-style-type: none"> • Oxygen delivery <ul style="list-style-type: none"> – Keep arterial oxygen saturation > 93~95% – Central venous oxygen <ul style="list-style-type: none"> • To assess balance between oxygen supply and demand • ScvO₂ >70% 	

三、上課人員簽到單

2016 Emergency training Course in Second General Hospital of Mongolia

Attendance sheet

No	Signature	No	Signature
29	BolorHtetseg.	44	N. Tuvshinbayar
30	N. Munkhulzii	45	Enkbat Nansalmaa
31	ts. Anydavi	46	Baatar Tungalag
32	ts. Mart	47	Tseren-achir Tserenchimed.
33	N. Urgamaltseseg.	48	Baatar Sukh Tsmirtseseg.
34	P. BAVARMAA	49	Davaasuren Uhzissai Khara.
35	G. Bandoimeg	50	Gankhuyag Gereltuya.
36	D. Tseveg	51	Munkhtagtokh Tuvshinbayar
37	A. Qunaa BAZAR	52	Bat-Erdene Mungunmunkh
38	P. Tungalag	53	
39	T. Nangilsuren	54	
40	YURA Nandintsetseg.	55	
41	Otgortseg Bolor Erdene	56	
42	Maikhaat Narantsetseg.	57	
43	Lamiav Enkh LARJAL	58	

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Attendance sheet

No	Signature	No	Signature
29	Nyanjav Byambatsetseg	44	Dashdemberel. Oyuantulkhuuz.
30	Dorjpalam Battssetseg	45	Myagmarjav Namhai
31	Dugarsuren Naranzul	46	Purevjav Gansukh
32	Damba Cevseg.	47	marat Bayar.
33	Uranbayar Bambald.	48	Bootar sven Boosanjargal.
34	Botgerel Sarantuya	49	Khurelbestar Munkhaya
35	Maint Naranzsetseg	50	baatar chuluun Bagtsetseg
36	Otgontugs Bolor-Erdene	51	Tseveenjav Anudari.
37	Rentsenbaymbaa Dolgormaa	52	Nergui Munkhulzii
38	Legtseg Dolgormaa	53	Natsagdorj Bayarjargal
39	Baasanjav "MUMS" Tolbayar	54	Dorjgotob Altansetseg
40	Lamjav. Enbjargal.	55	OuyngereL. Jashsetseren
41	Enkhtur Enkhzul	56	Undral Borkhuu,
42	egden togtoh Khishig batzar	57	Thimge Hongortul
43	OTGONJARGAL Nyantkhuv	58	Arinuzaga

59. Sebov Altanbayara

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Attendance sheet

15th December

No	Signature	No	Signature
1	Dr. Dolgorasuren	15	t.S. byambasuren
2	Nr. Byambasuren	16	N. monkhzaya
3	G. Munkh-Orgil	17	Ch. Saïntujs
4	D. Amarcel	18	G. Erdenechimeg
5	A. Tungalay	19	B. Ariunzaya
6	A. Javzankhuu	20	H. Khongonjal
7	B. Sarantuya	21	M. Tsetseged
8	D. Oyurechimeg	22	N. Boyarjargal
9	P. Gansakh	23	B. Undral
10	D. Altantsetseg	24	P. Buyantuy
11	D. Erdeneasuren	25	D. Setseg
12	S. Sainzaya	26	D. Oyuantukhuur
13	E. Nomin Erdene	27	ch. Oyundelger
14	B. Khangai	28	O. Aibolot

Lg. Z. Tuguldur

2016 Emergency training Course in Second General Hospital of Mongolia

Attendance sheet

No	Signature	No	Signature
29	Bolorhetseg.	44	N. Tuvshinbayar
30	N. Munkhulzi	45	Enkbat Nansalmaa
31	ts. Anydov	46	Baatar Tungalag
32	ts. Mart	47	Tseren-achir Tserenchimed.
33	N. Urgamaltsetseg.	48	Baatar Sukh Tamiitsetseg.
34	D. BAVARMOR	49	Dava suren Uzitsai Khara.
35	G. Bandoimeg	50	Gankhyag Gereltuya.
36	D. Tsetseg	51	Munkhtogtokh Tuvshinbayar
37	A. Qunaa BAZAR	52	Bat-Erdene Mungunmunkh
38	P. Tungalag	53	
39	T. Nangilsuren	54	
40	YURA Nandintsetseg.	55	
41	Otgovtsef Bolor-Erdene	56	
42	Maikhaat Narantsetseg.	57	
43	Lamiav Enkhlargal.	58	