

Hypertensive Crisis

Timothy E. Bunchman

Professor and Director

Pediatric Nephrology & Transplantation

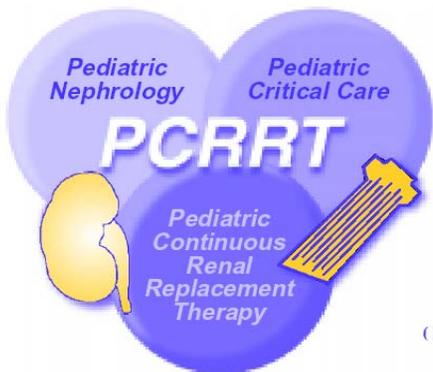
Children's Hospital of Richmond

Virginia Commonwealth Univ. School of
Medicine

Timothy.bunchman@vcuhealth.org

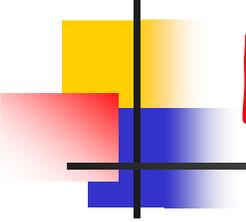
pedscrtr@gmail.com

www.pcrrt.com



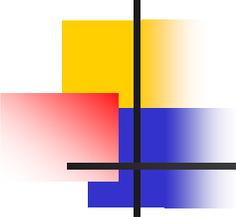
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Hypertension

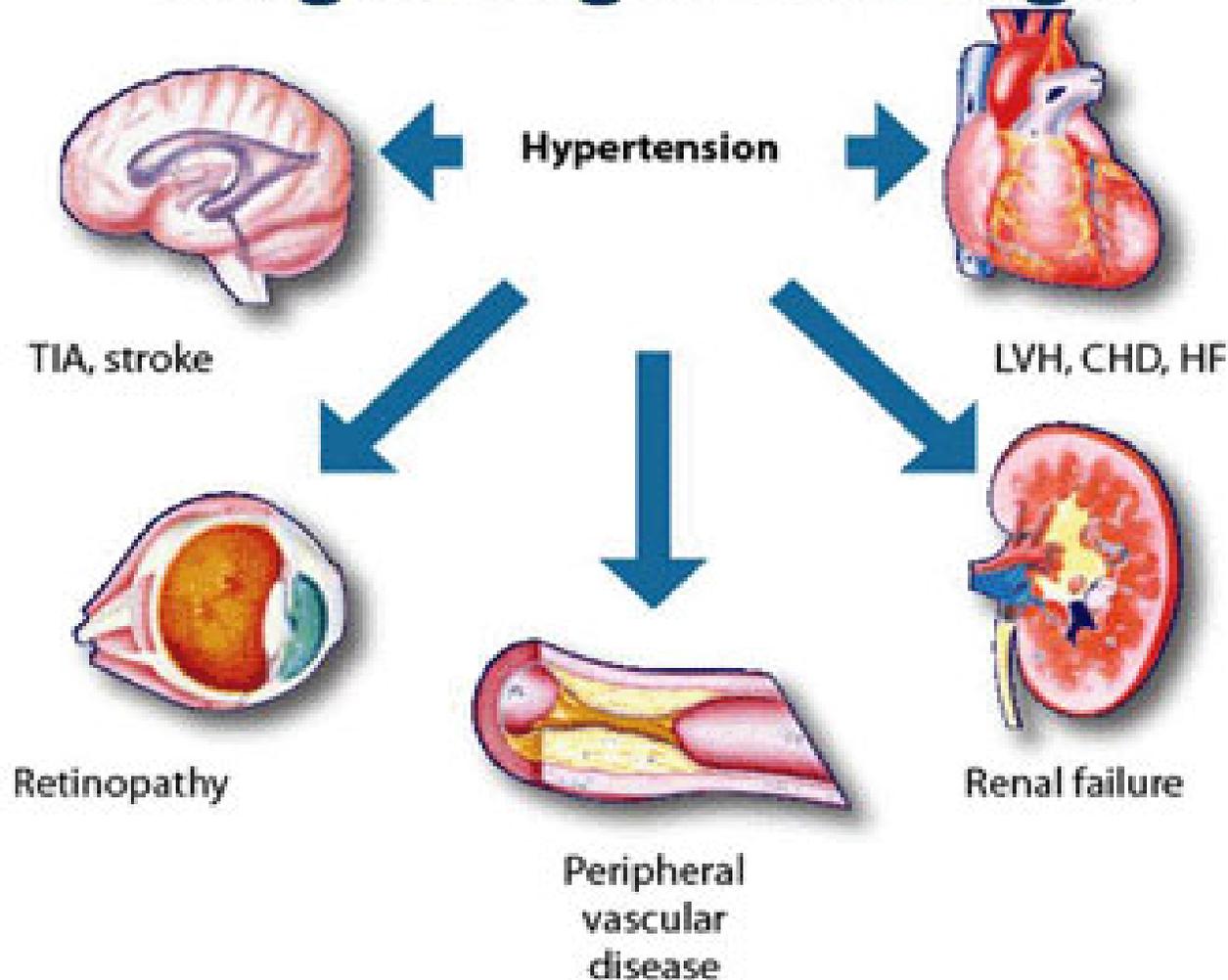
- Whether end organ symptoms or damage is present or not - is more important than the **absolute degree of BP elevation**
- The renin angiotensin system plays an important role in the genesis of hypertension crisis
- Important to assess whether the HT is chronic, acute, or acute on chronic



Definitions

- **SEVERE HYPERTENSION:** defined as 20 mm hg above the 95th centile
- **HYPERTENSIVE CRISIS :** defined as a sudden and abrupt elevation in blood pressure from baseline- generally 50% above normal- or >180/120
- **HYPERTENSIVE EMERGENCY** - a severe symptomatic elevation in BP with evidence of potentially life threatening symptoms or acute target organ damage
- **HYPERTENSIVE URGENCY** is severe hypertension with no target organ damage

Complications of Hypertension: Target-Organ Damage



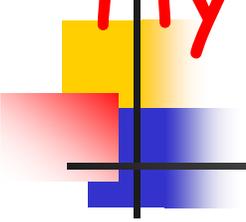
TIA, transient ischemic attack; LVH, left ventricular hypertrophy; CHD, coronary heart disease; HF, heart failure

Clinical evidence of End organ damage

- **CNS:** altered mental status-lethargy, coma, confusion, seizures, irritability, Facial N palsy, hemiplegia
(Exclude Head trauma or mass lesion)
- **Eye:** Papilloedema, retinal hemorrhages
- **Heart:** LVF, Pulmonary edema, S 3 gallop, new heart murmurs, LV hypertrophy
- **Renal:** Hematuria, proteinuria (Acute GN)



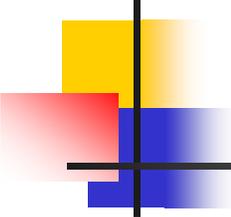
Hypertensive Encephalopathy



Most Emergencies manifest as ENCEPHALOPATHY
which is defined by

- Severe BP elevation with cerebral edema
- Neurological symptoms of lethargy, coma and seizures

Caused by cerebrovascular endothelial break down secondary to failure of cerebral autoregulation



Presentation to review

- Etiology
- Evaluation
- Treatment options

Neonatal Hypertension

- Renovascular disease
 - UA cath and TE
- Congenital renal anomalies
- Coarctation of Aorta
- Broncho pulmonary dysplasia
- CAH
- Renal vein thrombosis
- Renal parenchymal disease
- Iatrogenic Fluid overload
- ARPKD
- Tumours
- Hypercalemia

Childhood Hypertension

- Most often secondary to an underlying disease
 - Renal parenchymal disease: 60-70%
 - Ac GN, Reflux N
 - Renovascular disease : 5-25%
 - Renal A stenosis
- Coarctation of aorta - important cause in infancy
- Essential hypertension - emerging health concern in adolescents- **but rarely causes HT crisis**

HT in CKD patients-and Chronic Dialysis

- ECF expansion
- Salt intake
- Increased cardiac output and peripheral resistance
- High AT-II levels
- Sympathetic over activity
- Hyper PTH
- Uremic toxins
- Erythropoietin
- Non-compliance with antihypertensives
- Endothelial dependent Vd

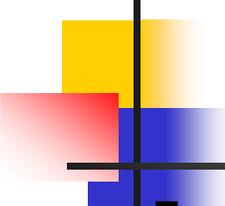
HTN post transplant pts

- Acute CNI toxicity
- TMA
- Effect of sirolimus
- High dose steroids
- Fluid overload
- Severe rejection and MA

Renovascular hypertension

RENAL ARTERY STENOSIS

- FMD, NF 1, TS
- Takayasu's arteritis
- Middle aorta syndrome
- William syndrome



Examination

- Anthropometry

- weight

- height

- BMI

- All peripheral pulses to be palpated

- Four limb BP

- Good general and systemic examination

- Neurocutaneous markers

- Peripheral edema

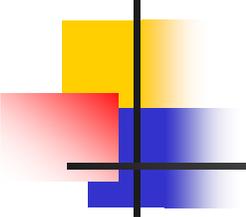
- Waist/Hip Ratio

- Waist circumference - Abdominal obesity

- ❖ Birth weight

- ❖ Post natal growth pattern

- ❖ High salt intake



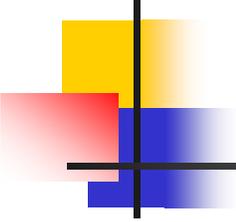
EVALUATION

Phase 1

CBC
Urinalysis
Urine Culture
Na/K/creat/BUN/
Ca/Uric acid
Lipid profile
Chest Xray
Renal USG,
Doppler
ECHO, ECG

Phase 2

Renal scan with ACE
inhibitor
Renin profile
MCU, DMSA
Urine catecholamines
Plasma and urine steroids



EVALUATION

Phase 3

Renal artery
imaging
Renal vein renin
sampling

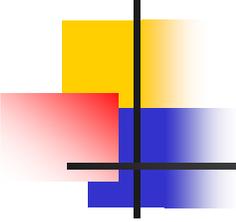
CO MORBIDITY

Fasting lipid profile
Fasting Glucose
Drug screen
Polysomnography

U/A

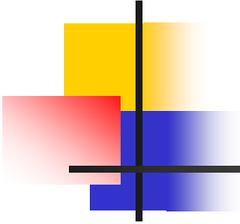
- Specific Gravity
- Proteinuria
 - Prot/Cr
 - UPEP
- + Hematuria + RBC
- + Hematuria – RBC
 - CPK
 - LDH



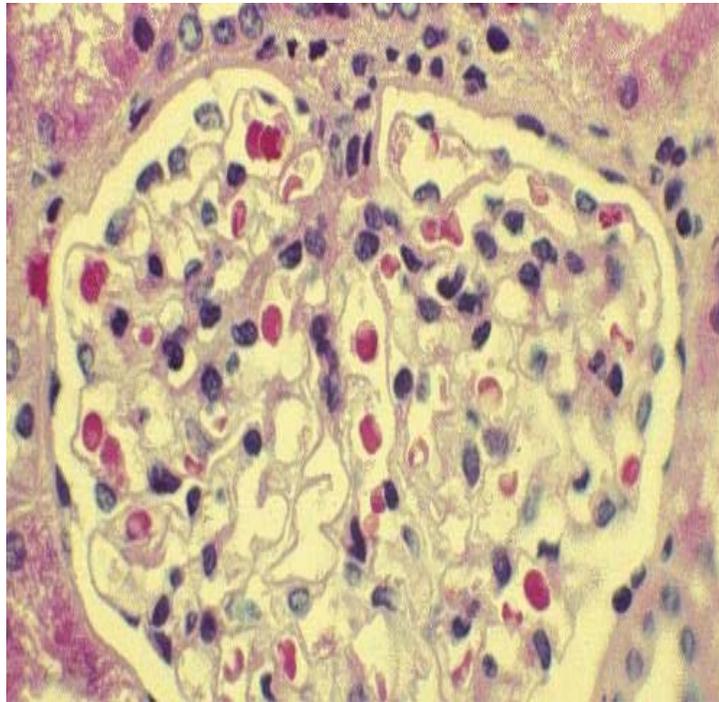


If

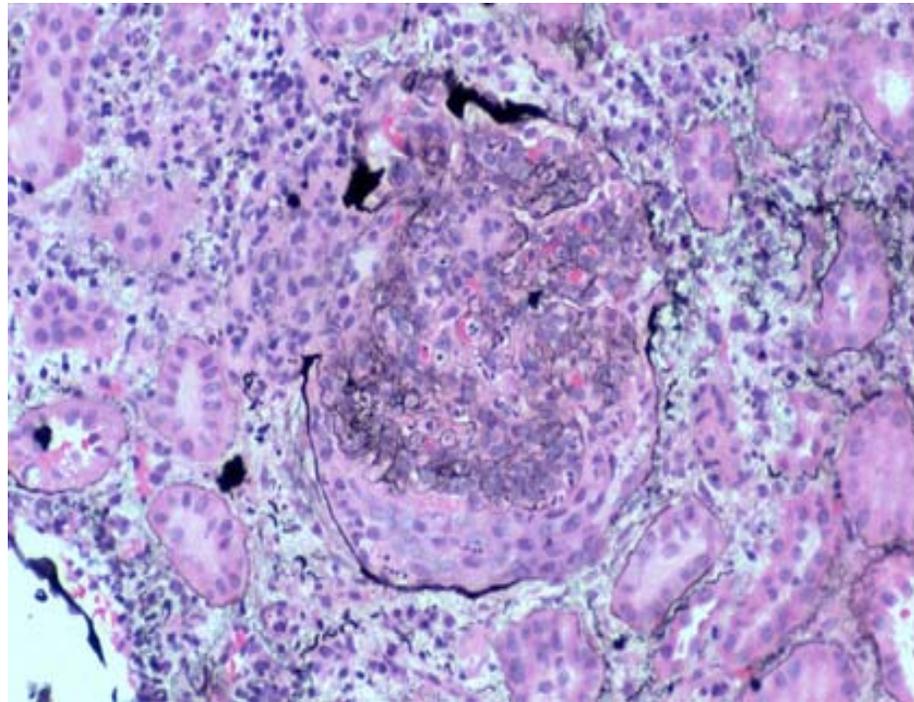
- Hypochloremic Metabolic Alkalosis
 - Adrenal induced Htn
 - RAS
- Renal insufficiency, low plasma proteins/albumin due to renal loss
 - bx
- Low complement
 - PIGN, SLE, MPGN may need to be bx

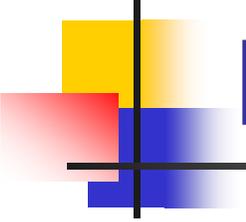


Normal



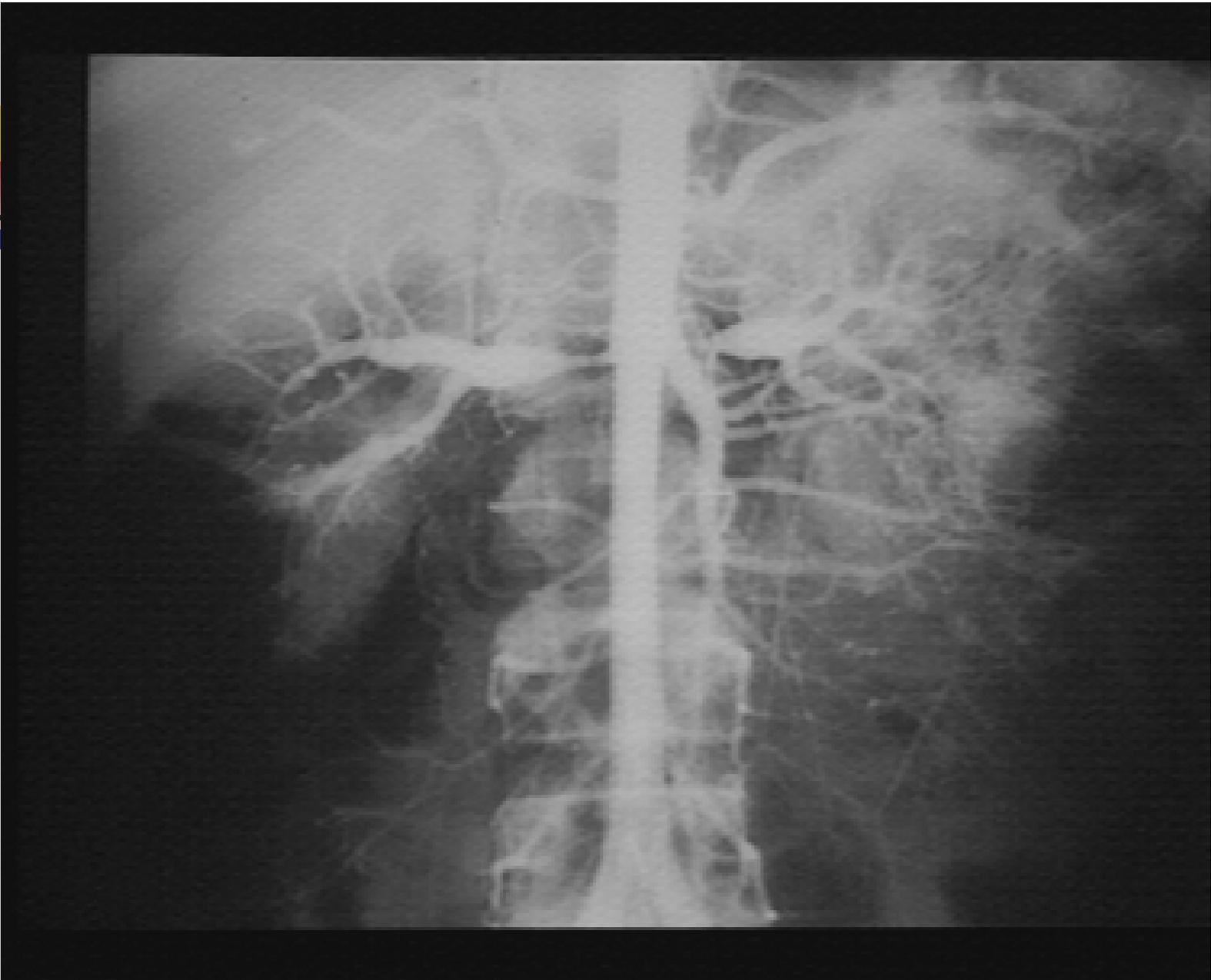
Crescentic GN

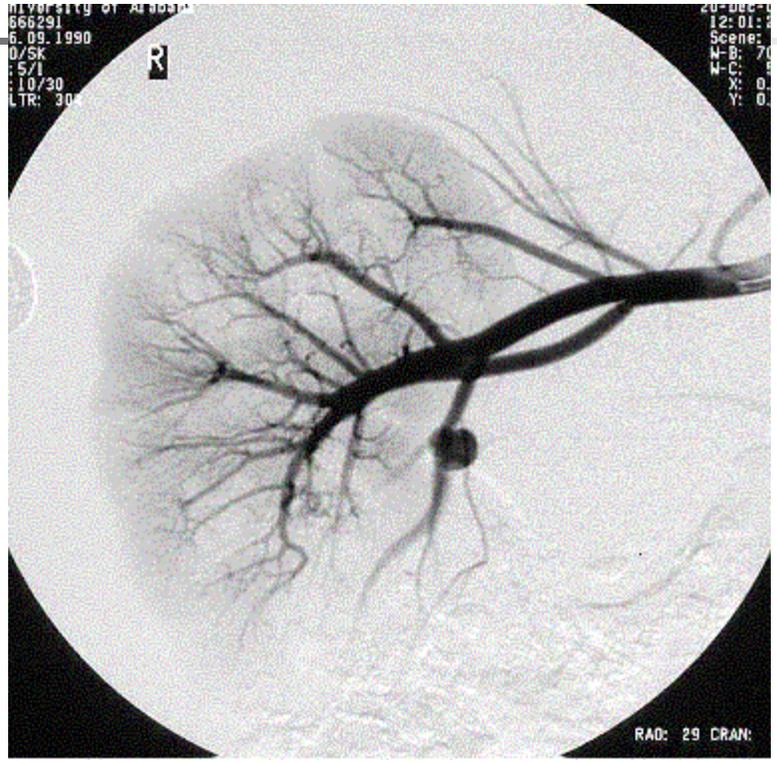
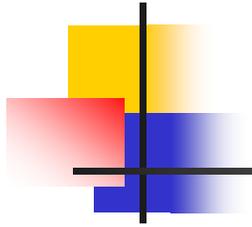


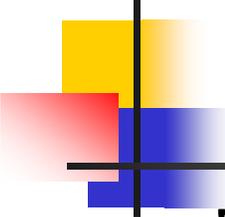


Renal imaging

- If there is a discrepancy in renal size
 - Angiogram with renal vein renins
 - Avoid use of diuretics, ACE, ARB or Beta blockers 1-2 weeks prior to this setting
 - DMSA scan for scar
 - Thin cut CT

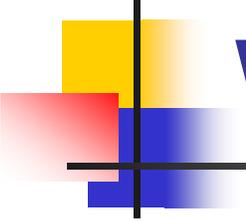






Renal Vein Renins

	High IVC 12 ng/ml/hr	
Rt Renal Vein 20 ng/ml/hr		Left Renal Vein 5 ng/ml/hr
	Low IVC 3 ng/ml/hr	



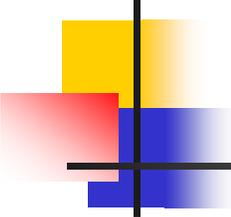
Why not draw ?

- Renins

- Random renins have not been found to be diagnostic...
 - effected by volume status, ACE inhibitors, ARBs, Beta blockers

- Aldosterone

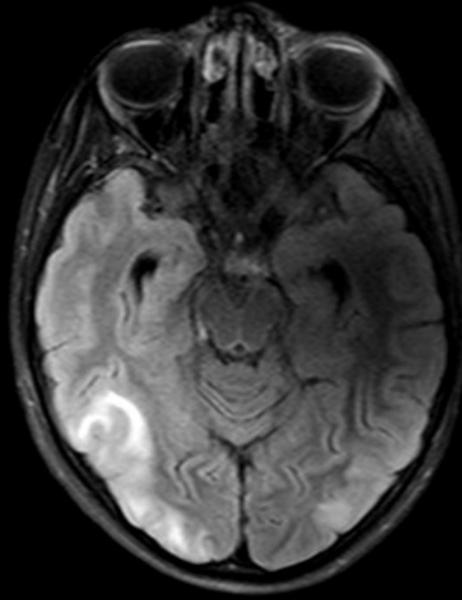
- May be helpful if evidence of a hypochloremic metabolic alkalosis



Case 1

- 6 years old male child with SRNS was diagnosed to have hypertension outside, started on Enalapril
 - Had one episode of change in is mental status
 - Intubated due to worsening of sensorium
 - He developed irrelevant speech and unable to bear weight after extubation
 - Referred here for further management.
- In casualty he had one episode of seizure for which he was started on antiepileptic
- EEG done was suggestive of bilateral epileptiform discharges
- MRI Brain with MRA was done

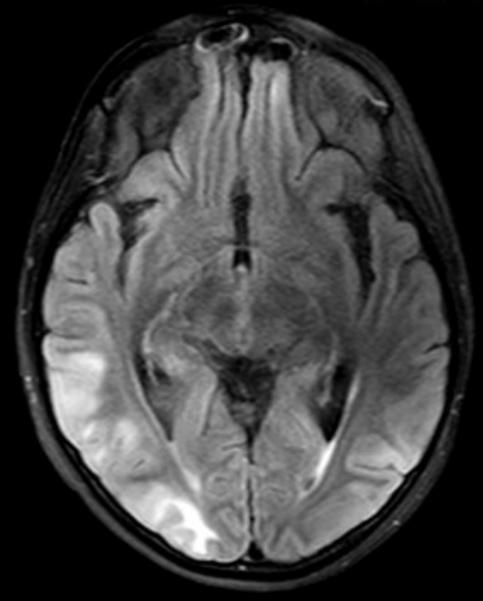
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Im:12
[AR]
Study Date:10/06/2015
Study Time:4:39:45 PM
MRN:



[LA] [RP]

C513
W892

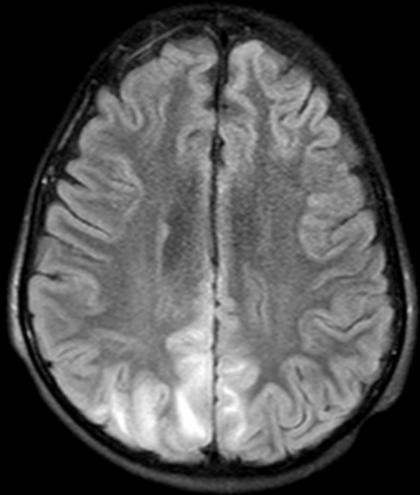
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Study Time:4:39:45 PM
MRN:



[LA]

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MRN:

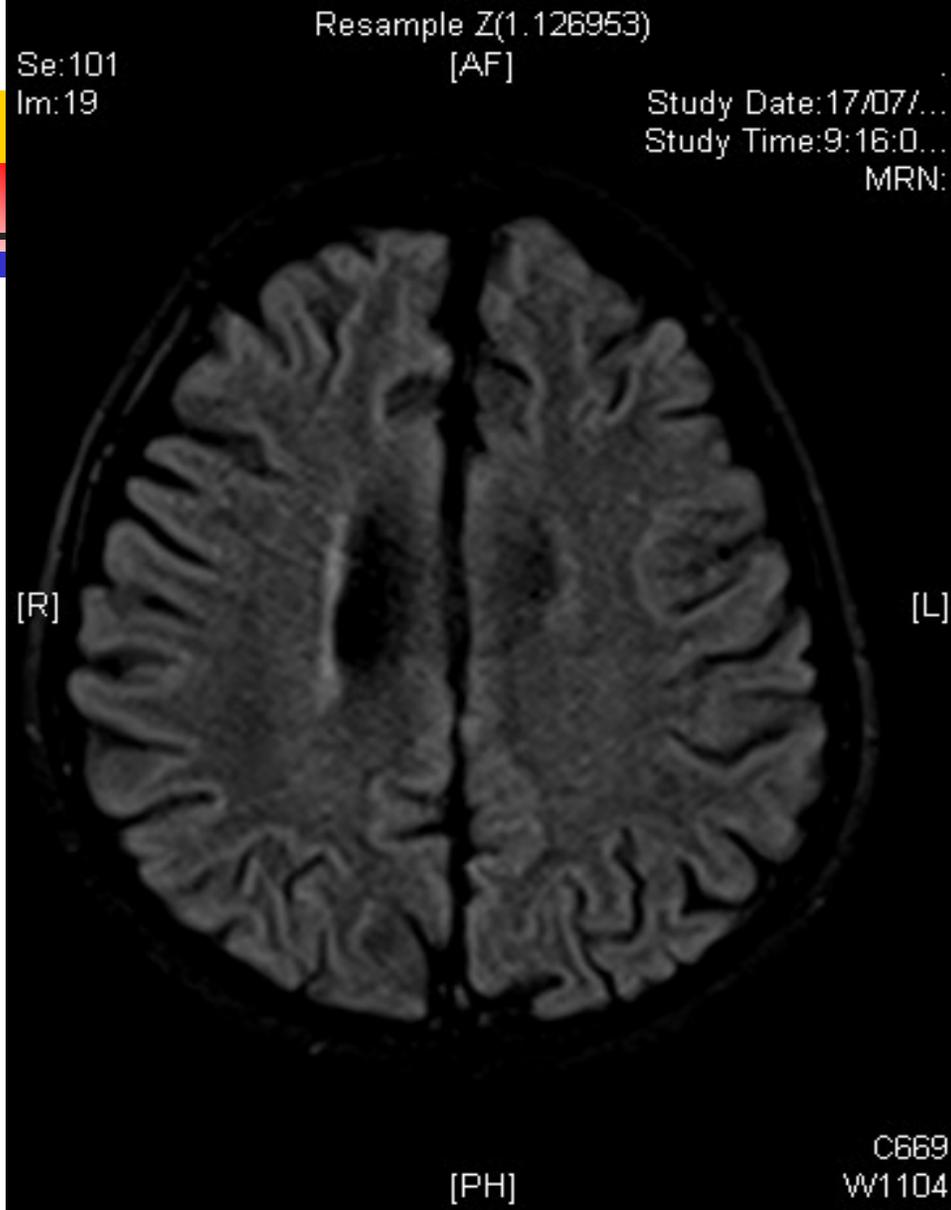


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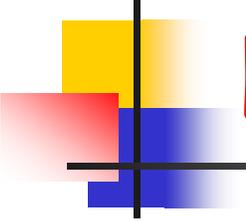
[LA]

C636
W1106

Bilateral asymmetric
gyral swelling and
hyperintensity and
symmetric posterior
thalamic
hyperintensities



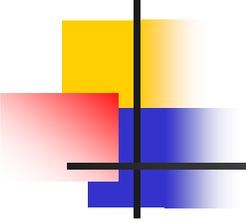
After a month -
resolution of
findings



DIAGNOSIS ???

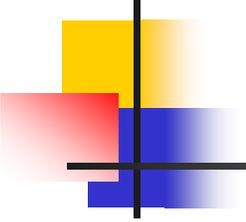
PRES

POSTERIOR REVERSIBLE
ENCEPHALOPATHY SYNDROME



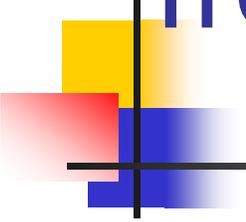
PRES

- Hypertensive encephalopathy occurs due to
 - Cerebral hyper perfusion
 - Endothelial dysfunction
 - Microvascular injury
 - Cerebral edema
- These manifests as PRES in imaging studies



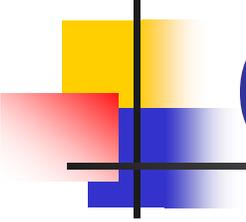
PRES

- Occurs due to vasogenic edema, onset of hypertension is acute
- Often a trigger in other conditions like
 - Treatment with immunosuppressive drugs
 - Vasculitis
 - Renal insufficiency
- Endothelial dysfunction and NO depletion are proposed mechanisms
- Symptoms
 - Altered mental status, Headache, Seizures



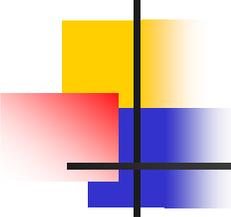
Treatment Options for acute HTN

- Angiotensin Receptor Blockade (ARB)-**avoid**
- Central Action
- Diuretics-avoid
- Vasodilators/alpha blockers
- Beta Blockers-**avoid if hyperkalemia**
- Calcium Channel blockers
- ACE inhibitors-**avoid**



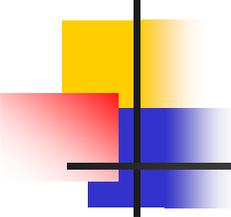
Angiotensin Receptor Blockade (ARB)

- Newer generation of class of drugs
- Not a role in the Rx of malignant Hypertension
- Used best in patients with CKD (chronic kidney disease)
- Avoid if CKD/AKI/hyperkalemia



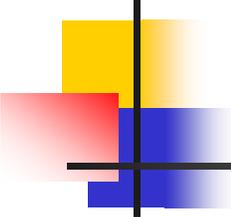
Central Action

- Not a role as a primary agent
- PO/Transdermal Clonidine good option for sedation withdrawal hypertension
- Problem with patch is at the time of hypotension a patch can be forgotten
- If on po Clonidine (data on patch less clear) and drug abruptly stopped, one is at risk for rebound significant hypertension



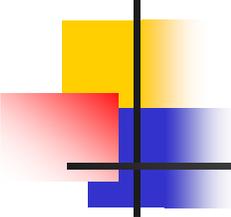
Diuretics

- Use as an adjunct to other agents only
- Not a role as a primary antihypertensive
- Side effects of electrolyte disturbances, elevated uric acid, lipids



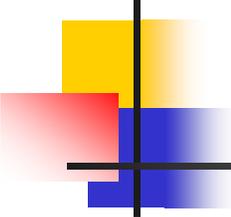
Beta Blockers

- Pure Beta blockers have limited role in hypertensive crisis
- Avoid if hyperkalemia, bradycardia



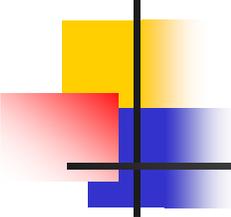
Beta Blockers

- Esmolol-good for tachycardia
 - Ld 100-500 mic/kg then 50-500 mic/kg/min
 - Wiest et al, J Thor Cardiovasc Surg 1998 4:890
- Labetalol (Alpha/Beta)
 - Ld 0.2-1 mg/kg then 0.5 – 2 mg/kg/hr
 - J Peds 1992 120:140
- Metoprolol infusions
 - 1-5 mics/kg/min to titrate to effect
 - Peds Nephrol 2017 32:2107-2113



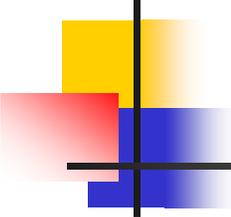
ACE inhibitors

- Should be limited as a primary agent due to the risk of
 - Influencing renin data if measured
 - Risk of AKI, hyperkalemia
 - Avoid in severe coarctation of the aorta or in bilateral renal artery stenosis due to risk of AKI



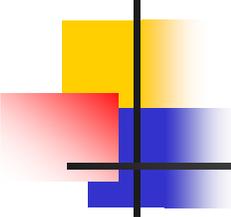
ACE inhibitors

- The only IV option is enalaprilat
 - 5-10 mic/kg/dose q 4-8 hrs
 - Wells et al, *Pediatr* 1988 113:403
- Preferred drug in micro embolic states with associated Htn
 - UAC induced
 - Renal vein thrombosis



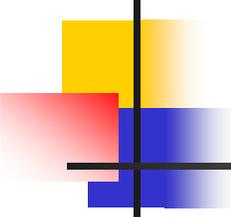
Vasodilators/alpha blockers

- Nitroprusside
 - Direct arterial vasodilator
 - Use with caution in AKI
 - Cyanide toxicity
 - Replaced by newer generation
- Nitroglycerin
 - Arterial and veno dilator
 - Intravenous or Transdermal (0.5-2 inches)



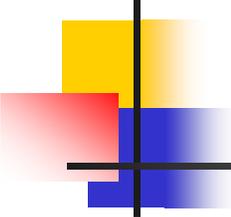
Vasodilators/alpha blockers

- Fenoldopam
 - Dilator with improvement in renal blood flow
 - 0.1 mic/kg/min to max 5 mic/kg/min
 - Stauser et al, AM J Ther 1999, 6:283
- Use in patients without Htn to enhance Renal Blood Flow and UO has not been well studied but may decrease BP to much to offset benefit of RBF preference



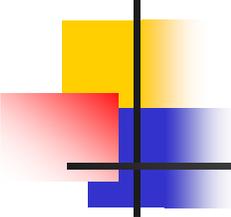
Vasodilators/alpha blockers

- Phentolamine
 - Used exclusively in Rx of Pheochromocytoma
 - 0.1-0.2 mg/kg bolus and q 2-4 hrs
 - Use preop to effect systemic symptoms of flushing, significant BP swings
- [Bholah R et al](#), Review of Pediatric Pheochromocytoma and Paraganglioma.
 - [Front Pediatr.](#) 2017 Jul 13;5:155. doi: 10.3389/fped.2017.00155. eCollection 2017.



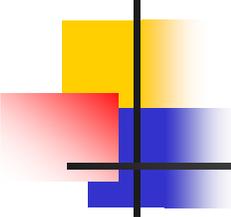
Calcium Channel Blockers

- Primary drug used for Malignant Htn
- Safe as an intravenous agent
- Class of drug of choice in patients on calcinurin inhibitors (tacro, csa, rapamycin)
- Nifedipine SL
- Nicardipine IV



SL Nifedipine

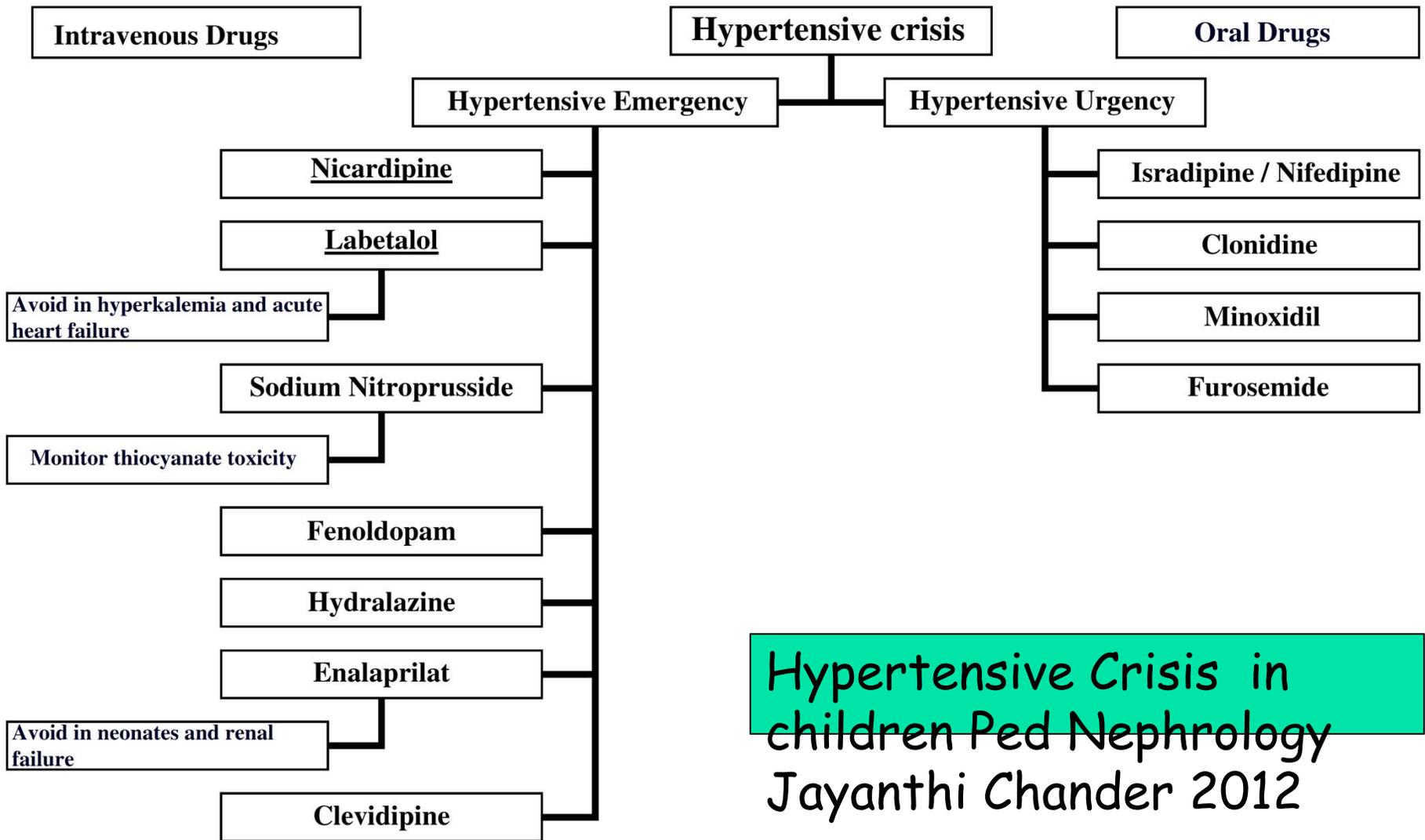
- Immediately effective but watch for rapid and worsening rebound
- No longer used in adult due to sudden death
- Leonard et al Ped Emg Med 2001 17:435
 - 2 children given SL due to Malignant Htn in the ED..both improved but within 30 mins both rebound and had strokes!



Nicardipine

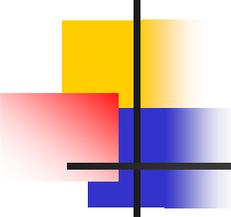
- Immediate on set of action
- Easy to titrate at bedside
 - 0.5-5 mic/kg/min as a continuous gtt
 - J Pediatr 2001 139:38
- Perhaps a role as an intermittent bolus
 - 10-20 mic/kg/dose IV/5 mins every 1-3 hrs

Algorithm for Treatment of Hypertensive Crisis



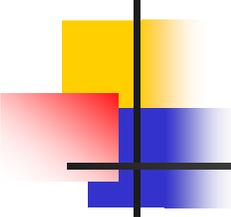
Hypertensive Crisis in children Ped Nephrology
 Jayanthi Chander 2012

Algorithm for treatment of hypertensive crisis



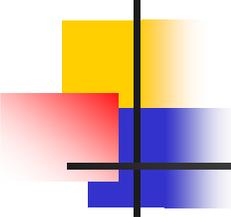
Treatment Strategy

- Like antibiotics decision making learn one from each class and stay with it
- If the goal is to get the patient improved but to get them to the ward
 - Begin oral, Transdermal therapy soon after beginning IV therapy if the patient can tolerate it



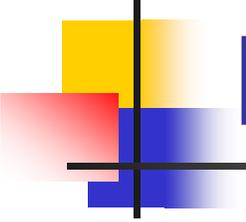
Drugs that are easy to use

- Consider CCB or vasodilators as the first line drugs for Htn for these have low risk profiles, less influence on interpretation of other data



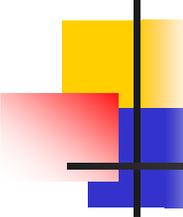
Thank you

- Timothy.bunchman@vcuhealth.org
- pedsrct@gmail.com
- www.pcrct.com
 - Contains all talks given at the PCRRT meetings from 2000 to the most recent in 2017 in Orlando



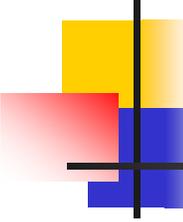
Signs and symptoms of hypertensive crisis includes

- seizures
- Heart failure
- Pulmonary edema
- All of the above



With a child has a bilateral renal artery stenosis with renal insufficiency then the intravenous drug to avoid is

1. IV Nicardipine
2. IV enalaprilat
3. IV atenolol
4. IV nitroglycerine



If a child has a seizure from a hypertensive crisis then the priority (of the first thing to do) is to

- Check a head CT scan
- Treat with seizure medications
- Bring the blood pressure down
- All of the above