VALIDITY OF THE ADJUNCTIVE USE OF BEDSIDE NONINVASIVE CLINICAL EXAMINATION AND TRANSCRANIAL DOPPLER ULTRASOUND IN OUTCOME PREDICTION AFTER CARDIAC ARREST

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ABSTRACT

Background: Neurological recovery after resuscitation from cardiac arrest (CA) can be potentially evaluated by clinical examination. The aim of the present study was to validate the adjunctive use of transcranial Doppler (TCD) ultrasound and the clinical examination for guiding outcome prediction within 72 hr after cardiopulmonary resuscitation (CPR). **Methods:** 45 adult patients (mean age 51 ±12 yr) successfully resuscitated from CA were prospectively included in this study. Clinical examination including (Glasgow coma scale (GCS)- motor score, pupil and corneal reflexes) were carried out after CPR and 24hr, 48hr and 72hr later. Mean cerebral blood flow velocity (MFV) and pulsatility index (PI) were assessed using TCD ultrasound at the same previous time points. The patients were followed up for 28 days and then were retrospectively classified into two groups according to cerebral performance category scale (CPC). Group I (CPC1-2) of good outcome and group II (CPC 3-5) of poor outcome. **Results:** 28.9% of patients developed good outcome (CPC1-2) while, 71.1% developed poor outcome (CPC3-5). The number of patients with GCS – motor score ≤ 3 or absent pupil or corneal reflexes was decreased over time until 72 hr in group I in comparison to group II. MFV values were low after CPR in both groups , but increased significantly in group I in comparison to group II over time until 72 hr. Also PI mean values were high after CPR in both groups. However, these values decreased significantly over time until 72 hr in group I in parallel to increase in MFV in comparison to group II. At 72hr after CPR, clinical examination (GCS - motor score ≤ 3, absent pupil and corneal reflexes) was +ve predictor of poor outcome of 92.6% with sensitivity of 78.1% , specificity of 84.6% and accuracy of 80%. TCD ultrasound measurement (MFV and PI) at 72 hr was +ve predictor of poor outcome of 96.4% with sensitivity of 84.4%, specificity of 92.3% and accuracy of 86.7%. The combination of clinical examination and TCD measurement raised the percentage of +ve prediction of poor outcome after 72 hr of CPR to 100% with sensitivity of 90.6%, specificity of 100% and accuracy of 93.3%. **Conclusion:** The adjunctive use of bedside, noninvasive clinical examinations and TCD ultrasound after 72 hr of CPR can potentially achieve more accurate prediction of poor outcome after CA rather than the use of single modality alone.

Keywords: clinical examination, transcranial Doppler, prediction, outcome, cardiac arrest

INTRODUCTION

Despite the advances in cardiopulmonary resuscitation (CPR) practices, the outcome of most cardiac arrest (CA) cases remains poor[(1)]. Patients successfully resuscitated from CA are at high risk of death or being neurologically devastated survivors.

Although numerous researches concerning outcome prediction after CA have been performed, limited degree of accuracy of early prognostication still exist[(2, 3)].

Several parameters (somatosensory evoked potential "SSEP", electroencephalography "EEG" and biochemical markers) have been studied to predict outcome after CA. SSEP is considered one of the most reliable prognostic tests[(1, 2)]. However, it requires advanced neurological training, can be interpreted only in specialized centers and is influenced by electric field in intensive care[(2)].

Burst suppression or generalized epileptiform waves on EEG have insufficient prognostic accuracy of predicting poor outcome[(1)]. Furthermore, the need for expertise and its susceptibility to effect of many drug and metabolic disorders may limit its use[(2, 4)].

Previous studies have investigated the usefulness of increased serum neuron-specific enolase (NSE) as a marker of brain damage and found that serum NSE levels > 33 ng/L at 72 hr after CPR can predict poor outcome. However, values below this cutoff level did not indicate good outcome[(5-7)]. Also, the NSE measurement is time consuming, not readily available, and not standardized[(1)].

These previous prognostic parameters may be sufficient but do not provide a good evaluation of cerebral blood flow changes after CPR. Knowledge of cerebral vascular circulation is fundamental for understanding the sequelae of CA. Transcranial Doppler ultrasound (TCD) records the velocity and pulsatility of cerebral blood flow, permitting frequent or continuous (beat to beat) cerebral hemodynamic analysis[(8)]. Previous human and experimental studies showed that decreased cerebral blood flow velocities and increased cerebral blood flow resistance in the early phase after CPR may be correlated with poor outcome[(7, 9-13)].

The clinical neurological examination is the widest applicable tool for prediction of outcome

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after CA , even in institutions with limited technologies and expertise[2, 14]. An evidence-based review[1] showed that absent pupil and corneal reflexes within days 1 to 3 after CPR or absent motor or extensor response to pain after three days of CPR can be strongly associated with poor outcome. However, there is limited certainty of clinical examination alone to predict survival after CPR[15], also the use of sedation in intubated patients may influence the findings[2] so, a multimodal prognostic approach may be essential[15,17].

The aim of the present study was to validate the adjunctive use of TCD ultrasound and the clinical examination for guiding outcome prediction within 72 hr after CPR.

PATIENTS AND METHODS

After approval of the hospital ethics committee, 45 adult patients (24 males, 21 females)with mean age of 51± 12 years were successfully resuscitated from cardiac arrest and survived for a minimum of 72 hr after CPR in the emergency and surgical ICU at Zagazig University hospitals were included prospectively in this study. A written informed consent was obtained from the nearest relatives of every patient.

Patients with head trauma . Preexisting intracranial lesion, known terminal illness before CA , or those in whom satisfactory TCD examination could not be performed were excluded from this study. All patients needed intubation and mechanical ventilation. Vasopressors were used to achieve a target of mean arterial blood pressure between 70 and 100 mmHg. Monitoring of arterial blood pressure ,arterial blood gases, SpO_2, body temperature and blood glucose levels was continued for 72 hr after CPR. Clinical examination was carried out after CPR , at 24 hr, 48 hr and 72 hr later (during the daily interruption of sedation in sedated patients). The clinical examinations including (Glasgow coma scale-motor score , pupil and corneal reflexes) were based on criteria in the Ann 2006 guidelines[1] and according to previous works of Levy et al.[14]. In assessing the motor response, a painful stimulus may be applied to the sternum (firm twisting pressure applied with the examiner's knuckles).The pupil reflex and corneal reflex were examined in both eyes . Also all patients were observed for epileptic attacks for 72 hr after CPR .

Mean cerebral blood flow velocity (MFV) and Pulsatility index (PI) were assessed using TCD ultrasound (multidop DWL) after CPR, 24 hr, 48 hr, and 72 hr later. The middle cerebral artery was insonated with 2 MHz emission frequency probe through a transtemporal window at depth of insonations varied from 35 to 55 mm as described by Asalid method[8]. The middle cerebral artery was chosen for the examination because this vessel perfuse about 80 % of the cerebral hemisphere and because of its ease accessibility for TCD. Arterial blood gases , mean arterial blood pressure and temperature were recorded at the same time with the TCD measurements.

Neurologic assessment was performed on 1st , 7 , 14 , 21 and 28 days after CPR using Pittsburgh cerebral performance category (CPC) scale (box)[18] according to 5 levels 1-5 that is commonly used in this setting.

At the end of the 28 days follow up period , retrospectively, the patients were divided into two groups according to clinical outcome using (CPC) scale : group I (CPC 1-2) : all patients who had a good or moderate neurological recovery i.e. survived with or without neurological disability. Group II (CPC 3-5) : all patients with poor outcome i.e. those who had severe neurological disability, developed persistent vegetative state or died.

Box. Glasgow-Pittsburgh Cerebral Performance Categories[18]

1. Good Cerebral Performance
Conscious: Alert, able to work and lead a normal life. May have minor psychological or neurological deficits (mild dysphasia, nonincapacitating hemiparesis, or minor cranial nerve abnormalities).

2. Moderate Cerebral Disability
Conscious. Sufficient cerebral function for part-time work in sheltered environment or independent activities of daily life (dressing, traveling by public transportation, and preparing food). May have hemiplegia, seizures, ataxia, dysarthria, dysphasia, or permanent memory or mental changes.

3. Severe Cerebral Disability
Conscious. Dependent on others for daily support because of impaired brain function (in an institution or at home with exceptional family effort). At least limited cognition. Includes a wide range of cerebral abnormalities from ambulatory with severe memory disturbance or dementia precluding independent existence to paralytic and able to communicate only with eyes, as in the locked-in syndrome.

4. Coma, Vegetative State
Not conscious. Unaware of surroundings, no cognition. No verbal or psychological interactions with environment.

5. Death
Certified brain dead or dead by traditional criteria.

STATISTICAL ANALYSIS

Data were checked, entered and analyzed by using SPSS version 15. Data were expressed as mean ±SD for quantitative variables, number and percentage for qualitative variables. Chi-squared (X^2) and t test were used when appropriate. Odd's ratio and 95% confidence interval (CI) were measured to quantify the risk of poor outcome .Validation of clinical examinations and TCD for prediction of outcome were assessed by calculation of sensitivity , specificity , + ve predictive value and – ve predictive value. P-value <0.05 was considered statistically significant.

RESULTS

This study included 45 successfully resuscitated adult patients who suffered from CA. Within the 28 days follow up period, 13 patients (28.9%) developed good outcome (group I) according to CPC scale, of whom 4 patients had CPC level of 1 and 9 patients had CPC level of 2. The remaining 32 (71.1%) patients (group II) developed poor outcome, of whom 3 patients had CPC level of 3 and 6 patients had CPC level of 4, while 23 patients died (CPC level of 5).

There was no significant difference in the mean age of the patients of group I (52±11 yr) and that of patients of group II (49±13 yr) P=0.46.

10/13 (76.9%) patients of group I had VF/pulseless VT as the most frequently observed rhythm before CPR versus 13/32 (40.6%) patients in group II (P=0.04). While asystole or pulseless electric activity were the most observed rhythm in group II 19/32 (59.4%) versus 3/13 (23.1%) patients in group I (P=0.04). These differences were statistically significant.

There was no significant difference in the mean duration of CPR between the two groups (13.5 ± 10.4 min in group I versus 17.1 ± 11.6 min in group II) P=0.32. However, patients of group I had highly significantly shorter duration of mechanical ventilation (2.5±2.1 days) than in patients of group II (11.2±7.5 days) P<0.001.

Similarly, the duration of unconsciousness was highly significantly shorter in patients of group I (3.3±2.8 days) than in group II (14.5±10.7 days) (P<0.001).

Seizures were seen within 72hr after CPR in 15/32 (46.9%) patients of group II, while were seen in only 2 (15.4%) patients of group I. This difference between the two groups was statistically significant P=0.04 (Table 1). No significant differences were found between group I and group II after CPR or at 24hr, 48hr, and 72hr regarding mean arterial blood pressure (MAP), arterial PCO_2 and temperature (table 2).

Clinical Examination:

The number of patients with GCS - motor score ≤ 3 or absent pupil and corneal reflexes was decreased over time until 72 hr in group I in comparison to group II (table 3).

After 72 hr of CPR 21/32 (65.6%) patients of group II had GCS - motor score ≤ 3, while only 3/13 (23.1%) patients in group I had GCS - motor score ≤3. This difference was statistically significant (P= 0.009). GCS - motor score ≤3 was associated with 6.36 fold risk rise of poor outcome (95% CI 1.22 - 37.31).

In group II we observed 25/32 (78.1%) patients had absent pupil reflexes, while only 1/13 (7.7%) patient in group I had absent pupil reflexes

at 72 hr after CPR. This difference was highly significant (P < 0.001). Absent pupil reflex was associated with 42.86 fold risk rise of poor outcome (95% CI 4.27- 1050.3).

In addition , absent corneal reflexes at 72hr were observed in 27/32 (84.4%) patients of group II versus no one in group I. This difference was highly significant (P < 0.001).

TCD Measurement:

Analysis of TCD measurement (table 4) showed that the MFV of the middle cerebral arteries were low in both groups after CPR (37±4cm/s in group I and 35±5 cm/s in group II) P= 0.2.

During the following 72 hr MFV mean values were significantly higher in group I than in group II as the following : at 24hr the MFV mean values were 58±8 versus 49±8 cm/s (P=0.0013), at 48hr 60±10 versus 46±6cm/s (P=0.04) and at 72hr 61±12 versus 47±11 cm/s (P=0.001) respectively.

After CPR , PI mean values were 1.3± 0.2 in group I and 1.4 ± 0.2 in group II; however, PI appeared to be decreased in parallel to the increase in MFV. There were significantly lower mean values of PI in group I than in group II during the following 72 hr { at 24hr 1.1±0.1 versus 1.3±0.2(P=0.04), at 48 hr 1.0±0.09 versus 1.24±0.1 (P < 0.001) and at 72hr 0.9±0.1 versus 1.21±0.2 (P= 0.012) } respectively.

Combination of clinical examination and TCD ultrasound in prediction of outcome at 72hr after CPR:

The clinical examination including (GCS-motor score ≤3, absent pupil reflex, and absent corneal reflex) was positive predictor of poor outcome of 92.6% with sensitivity of 78.1%, specificity of 84.6% and accuracy of 80%. TCD measurement (MFV and PI) was positive predictor of poor outcome of 96.4% with sensitivity of 84.4%, specificity of 92.3% and accuracy of 86.7%.

The combination of clinical examinations and TCD ultrasound raised the percentage of positive prediction to 100% with sensitivity of 90.6%, specificity of 100% and accuracy of 93.3%.

Table (1): Patients characteristics

	Group I (CPC1-2) n=13		Group II (CPC3-5) n=32		P value
Age (years)	52±11		49±13		0.46
First cardiac rhythm					
VF/Pulseless VT	10	(76.9%)	13	(40.6%)	0.04*
Asystole/pulseless electric activity	3	(23.1%)	19	(59.4%)	0.04*
Duration of CPR (min)	13.5±10.4		17.1±11.6		0.32
Duration of unconsciousness (days)	3.3±2.8		14.5±10.7		0.001**
Duration of mechanical ventilation (days)	2.5±2.1		11.2±7.5		0.001**
Seizures present	2	(15.4%)	15	(46.9%)	0.04*

CPR : Cardiopulmonary resuscitation
VF*: Ventricular fibrillation*
VT : Ventricular tachycardia
* P < 0.05 significant
** P < 0.001 Highly significant

Table (2): Changes in MAP, temperature and $PaCO_2$ during the first 72 hours after CA

		Group I (n=13)	Group II (n=32)	P-value
MAP (mmHg)				
	After CPR	79±9	82±10	0.35
	24hr	81±10	80±9	0.74
	48hr	78±7	79±8	0.69
	72hr	83±8	81±11	0.55
Temperature (C^o)				
	After CPR	36.41±0.3	36.5±0.1	0.29
	24hr	37.2±1.4	36.9±1.3	0.49
	48hr	38.1±0.4	37.6±0.7	0.09
	72hr	37.6±0.6	37.9±0.5	0.09
$PaCo_2$ (mmHg)				
	After CPR	41±4	43±3	0.07
	24hr	37±5	38±6	0.59
	48hr	38±3	37±5	0.5
	72hr	37±2	36±3	0.27

MAP: mean arterial pressure.
CPR: Cardiopulmonary resuscitation
$PaCO_2$: Arterial carbon dioxide tension

Table (3): GCS-motor score, pupil reflex and corneal reflex during first 72 hours after CA

	Group I (N=13)		Group II (N=32)		OR(95% CI)	P-value
GCS motor score						
≤3	9/13	(69.2%)	28/32	(87.5%)	3.11(0.51-19.67)	0.2
After CPR	7/13	(53.8%)	25/32	(78.1%)	3.06(0.64-15.2)	0.14
24hr	6/13	(46.1%)	22/32	(68.75%)	3.3(0.78-14.5)	0.06
48hr	3/13	(23.1%)	21/32	(65.6%)	6.36(1.22-37.31)	0.009*
72hr						
Absent pupil reflex						
After CPR	10/13	(76.9%)	30/32	(93.75%)	4.5(0.5-46.57)	0.13
24hr	8/13	(61.5%)	28/32	(87.5%)	4.38(0.763-26.69)	0.09
48hr	4/13	(30.8%)	27/32	(84.4%)	10.8(1.9-69.83)	0.002*
72hr	1/13	(7.7%)	25/32	(78.1%)	42.86(4.27-1050.3)	0.001**
Absent corneal reflex	10/13	(76.9%)	31/32	(96.9%)	9.3(0.71-262.29)	0.06
After CPR	7/13	(53.8%)	28/32	(87.5%)	6.0(1.07-36.33)	0.02*
24hr	3/13	(23.1%)	28/32	(87.5%)	23.3(3.5-187.74)	0.001**
48hr	0/13	(0.0%)	27/32	(84.4%)	-	0.001**
72hr						

CA: Cardiac Arrest
GCS: Glasgow Coma Scale
CPR: Cardiopulmonary Resuscitation

Table (4): Changes in mean flow velocity and pulsatility index during first 72 hours after CA

	Group I	Group II	P value
MFV (cm/s)			
After CPR	37±4	35±5	0.2
24hr	58±8	49±8	0.0013**
48hr	60±10	46±6	0.04*
72hr	61±12	47±11	0.001**
PI			
After CPR	1.3±0.2	1.4±0.2	0.13
24hr	1.1±0.1	1.3±0.2	0.04*
48hr	1.0±0.09	1.24±0.1	0.001**
72hr	0.9±0.1	1.21±0.2	0.012*

MFV: mean flow velocity
PI: pulsatility index
CA: cardiac arrest
CPR: Cardiopulmonary resuscitation

Table (5): Validity of clinical examination and TCD measurement in prediction of poor outcome after 72 hours of CPR

	Sensitivity %	Specificity %	Predictive value +ve %	Predictive value -ve %	Accuracy %
Clinical examination	78.1	84.6	92.6	61.1	80.0
TCD measurement	84.4	92.3	96.4	70.6	86.7
Combination of both	90.6	100.0	100.0	81.3	93.3

CPR: Cardiopulmonary resuscitation
TCD: Transcranial Doppler

DISCUSSION

Prediction of outcome after CA is a source of major interest between ICU physicians and family members[19]. As acute care resources have become greatly scarce and costly, identifying parameters that reliably predict outcome is essential. Results of the present study showed that asystole or pulseless electric activity were the most frequent rhythm causing CA in patients of group II who developed poor outcome than in patients of group I who developed good outcome. These results are in agreement with that of Pfeifer et al.[20]. However, others reported that asystole as primary cardiac dysrhythmia would be independent prognostic marker[21]. An evidence-based review showed that the type of cardiac arrhythmia may be related to poor outcome but does not discriminate accurately between patients with good or poor outcome[1].

In our study, the duration of unconsciousness was highly significantly shorter in patients of group I than in patients of group II ($p < 0.001$). This result is consistent with those of previous studies who showed a correlation between duration of unconsciousness for more than 2 days after CPR and poor neurological outcome[20, 22, 23]. Seizures may contribute to mortality and morbidity even in patients with intact brainstem reflexes or some motor responses [1].

In our study, the number of patients who had seizures within 72 hr after CPR was significantly higher in group II than in group I ($p = 0.04$).

Previous studies[7, 24, 25] showed that seizures can portend poor outcome. Nevertheless, none of the studies concluded that seizures accurately predict outcome. An evidence-based review[1] reported that only patients with myoclonus epilepticus during the 1st 24 hr after CPR have poor prognosis.

Clinical examination has the advantage of being greatly useful in the common clinical scenario because of its universal availability and ease of performance[2, 14, 26]. GCS monitoring is a valuable integrant in a multimodal prognostication approach[20, 27]. However, the motor part of GCS is more useful and accurate than GCS Sum score[14].

Previous researches[1, 14, 15, 26] recommended attention to the usefulness of pupil and corneal reflexes, and GCS- motor score in determining prognosis after CA.

In this study, after 72 hr of CPR, the clinical examination including(GCS-motor score ≤ 3 and

absent pupil and corneal reflexes) was positive predictor of poor outcome of 92.6% with sensitivity of 87.1%, specificity of 84.6% and accuracy of 80%. Practice Guidelines of the Quality Standards Subcommittee of the American Academy of Neurology[(1)] reported that no false prediction of poor outcome occurred after 72 hr of CPR for GCS-motor score ≤ 2 or ≤ 3. Similarly, no false prediction occurred for absent pupil and corneal reflexes after 24 hr to 72 hr of CPR. On the other hand, a more recent study by Rittenberger et al.[(15)] showed that a lack of pupil and corneal reflexes at 72 hr is highly predictive of poor outcome. However, non of the clinical examination tests were 100% predictive of death after 24 hr of CPR and so the authors suggested that clinical examination has insufficient prediction of outcome after 24 hr of CPR. Also, GCS-motor score ≤ 2 or ≤ 3 after 72 hr of CPR did not exclude survival and so the authors suggested that the motor examination is less useful than reported in the practice guidelines.

TCD ultrasound, an easily applicable, noninvasive, bedside technique, provides real-time information about changes in blood flow velocity in the main cerebral arteries which reflect the changes in cerebral blood flow (CBF) in patients undergoing CPR and also after Return Of Spontaneous Circulation (ROSC)[(10, 28)].

A recent review[(29)] showed that in patients who remain comatose 2 hr after ROSC, the predominant TCD pattern includes low MFV and high PI. Normal values should be reached after 72 hr in the absence of complications. If a hypodynamic pattern persists, this will be an indicator of poor outcome.

Edrgren et al.[(30)] studied CBF by positron emission tomography at 1st, 3rd, and 7th days after CPR in seven comatose patients and found a low CBF in the early days after CPR which increased later only in the survivors.

The pathogenesis of the abnormalities in CBF that occurred after CPR are not fully understood. Several suggested possibilities are vasospasm, or cerebral oedema and blood cell aggregation[(31, 32)].

An experimental study[(33)] showed that cerebral ischemia in a murine CA model which caused by abnormal vasoconstriction can be reversed by BQ_{123} (an endothelin type-A antagonist). In human, cerebral hypoperfusion caused by active cerebral vasoconstriction appeared to be caused by imbalance between local vasodilators and local vasoconstrictors[(34)].

In our study, the MFV mean values were low after CPR in both groups, which increased significantly in group I in comparison to group II over time until 72 hr after CPR. Conversely, PI mean values were high after CPR which decreased significantly in group I in comparison to group II over time until 72 hr after CPR. TCD measurement (MFV and PI) after 72 hr of CPR was positive predictor of poor outcome of 96.4 with sensitivity of 84.4%, specificity of 92.3%, and accuracy of 86.7%.

Our results are in agreement with that of Wessels et al.[(7)] who studied 39 patients undergone CPR and observed that peak systolic flow velocities (SFV) in MCA were significantly higher in group 1 (survivors) at four and 72 hr after CPR than in group 2 (non-survivors). Also, the Resistance Index (RI) in MCA tended to be lower in survivors group during the whole 72 hr. However, this difference was significant only at 8 hr after CPR.

Of note, unlike our results, in their study , systolic and diastolic velocities 1.5 hr after CPR appeared to be higher in patients who died than in survivors. The authors explained that by the necessity of high dose of administration of epinephrine following CPR.

On the other hand, Lemiale et al.[(12)] studied 18 out of hospital cardiac arrest patients and observed that at admission MFV were impaired in all patients, but reached normal values after 72 hr. Also, PI at admission were high and reached normal values after 72 hr. Interestingly, no significant differences were found between survivors and non-survivors regarding MFV and PI. The authors explained this by the use of therapeutic hypothermia during the 1st 24 hr after CPR which may alter change in CBF. Also, our results are in agreement with Feldges and Mehdorn[(9)] who investigated 150 comatose patients by serial TCD examinations and revealed that PI is considered a significant prognostic parameter.

Finally, our data revealed that the combination of clinical examination and TCD measurement after 72 hr of CPR raised the percentage of positive prediction of poor outcome to 100%, with sensitivity of 90.6%, specificity of 100% and accuracy of 93.3% than the use of single modality alone.

In conclusion, the adjunctive use of bedside noninvasive clinical examination and TCD ultrasound after 72 hr of CPR can potentially achieve more accurate prediction of poor outcome after CA rather than the use of single modality alone.

REFERENCES

1- Wijdicks EF, Hijdra A, Young GB, Basseti CL and Wiebe S. Practice Parameter. Prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): Report of

the Quality Standards Subcommittee of American Academy of Neurology. Neurology 2006; 67(2): 203-10.

2- Nolan JP, Neumar RW, Adrie C, Aibiki M, Berg RA, et al. Post-cardiac arrest syndrome: Epidemiology, pathophysiology, treatment and prognostication. A scientific statement from the International Liaison Committee on Resuscitation, the American Heart Association Emergency Cardiovascular Care Committee; the Council on Cardiovascular Surgery and Anesthesia; the Council on Cardiopulmonary, Perioperative, and Critical Care; the Council on Clinical Cardiology and the Council on Stroke. Resuscitation 2008; 79(3): 350-79.

3- Mayer SA. Outcome prediction after cardiac arrest: New game, new rules. Neurology 2011; 77(7): 614-5.

4- Scollo-Lavizzri G, Bassetti C. Prognostic value of EEG in post-anoxic coma after cardiac arrest. Eur Neurol 1987; 26: 161-70.

5- Fogel W, Krieger D, Veith M, Adam HP, Hund E, et al. Serum neuron-specific enolase as early predictor of outcome after cardiac arrest. Crit Care Med 1997; 25(7): 1133-8.

6- Almaraze AC, Borbrow BJ, Wingerchuk DM, Wellik KE, Demaerschalk BM. Serum neuron-specific enolase to predict neurological outcome after cardiopulmonary resuscitation: a critically appraised topic. Neurologist 2009; 15(1): 44-8.

7- Wessels T, Harrer JU, Jacke C, Janssens U, Klotzsch C. The prognostic value of early transcranial Doppler ultrasound following cardiopulmonary resuscitation. Ultrasound Med & Biol 2006; 32(12): 1845-51.

8- Aaslid R, Markwalder TM , Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. J Neurosurg 1982; 57(6): 769-74.

9- Feldges A and Mehdorn HM. The use of transcranial Doppler ultrasonography in a neurosurgical intensive care unit: Intracranial pressure and intracranial circulatory arrest. Wien Med Wochenschr 1990; 140: 567-70.

10- Lwis LM, Stothert Jr JC, Gomez CR, Ruoff BE, Hall IS, et al. A noninvasive method for monitoring cerebral perfusion during cardiopulmonary resuscitation. J Crit Care 1994; 9(3): 169-74.

11- Lwis LM, Gomez CR, Ruoff BE, Gomez SM, Hall IS, et al. Transcranial Doppler determination of cerebral perfusion in patients undergoing CPR: Methodology and preliminary findings. Ann Emerg Med 1990; 19(10): 1148-51.

12- Lemiale V, Huet O, Vigue B, Mathonnet A, Spaulding C, et al. Changes in cerebral blood flow and oxygen extraction during post-resuscitation syndrome. Resuscitation 2008; 76(1) 17-24.

13- Fukushima U, Sasaki S, Okano S, Oyamada T, Yochikawa T, et al. Noninvasive diagnosis of ischemic brain damage after cardiopulmonary resuscitation in dogs by using transcranial Doppler ultrasonography. Vet Radiol Ultrasound 2000; 41(2): 172-7.

14- Levy DE, Caronna JJ, Singer BH, Lapinski RH, Frydman H, et al. Predicting outcome from hypoxic-ischemic coma. JAMA 1985; 253(10): 1420-6.

15- Rittenberger JC, Sangl J, Wheeler M, Guyette FX, Callaway CW. Association between clinical examination and outcome after cardiac arrest. Resuscitation 2010; 81(9): 1128-32.

16- Young GB. Clinical practice, neurologic prognosis after cardiac arrest. N Engl J Med 2009; 361(6): 605-11.

17- Al-Thonayan E, Savard M, Sharpe M, Norton L, Young B. Predictors of poor neurologic outcome after induced mild hypothermia following cardiac arrest. Neurology 2008; 71(19): 1535-7.

18- Cummins RO, Chamberlain DA, Abramsan NS, Allen M, Baskett PJ, et al. Recommended Guidelines for Uniform Reporting of Data From Out-of-Hospital Cardiac Arrest: The Utstein Style. Circulation 1991; 84: 960-75.

19- Marion DW. Coma due to cardiac arrest: Prognosis and contemporary treatment. F-1000 Med Res 2009; 1: 89.

20- Pfeifer R, Borner A, Krack A, Sigusch HH, Surber R, et al. Outcome after cardiac arrest: Predictive values and limitations of the neuroproteins neuron-specific enolase and protein S-100 and the Glasgow Coma Scale. Resuscitation 2005; 65(1): 49-55.

21- Herlitz J, Ekstrom L, Wennerblom B, Axelsson A, Bang A, et al. Predictors of early and late survival after out-of-hospital cardiac arrest in which asystole was the first recorded arrhythmia on scene. Resuscitation 1994; 28(1): 27-36.

22- Snyder BD, Ramirez-Lassepas M and Lippert DM. Neurologic status and prognosis after cardiopulmonary arrest: I. A retrospective study. Neurology 1977; 27(9): 807-11.

23- Bates D, Carrona JJ, Cartlidge NE, Knill-Jones RP, Levy DE, et al. A prospective study of nontraumatic coma: methods and results in 310 patients. Ann Neurol 1977; 2(3): 211-20.

24- Van Cott AC, Blatt I, Brenner RP. Stimulus-sensitive seizures in postanoxic coma. Epilepsia 1996; 37(9): 868-74.

25- Young GB, Gilbert JJ, Zochodne DW. The significance of myoclonic status epilepticus in postanoxic coma. Neurology 1990; 40(12): 1843-8.

26- Booth CM, Boone RH, Tomlinson G , Desky AS. Is patient dead, vegetative, or severely neurologically impaired? Assessing outcome for comatose survivors of cardiac arrest. JAMA 2004; 291(7): 870-9.

27- Bassetti C, Bomio F, Mathis J , Hess CW. Early prognosis in coma after cardiac arrest: A prospective clinical, electrophysiological, and biochemical study of 60 patients. J Neurol, Neurosurg, Psychiat. 1996; 61(6): 610-5.

28- Polito A, Ricci Z, Chiara L, Giorni C, Lacoella C, et al. Cerebral blood flow during cardiopulmonary bypass in pediatric cardiac surgery: the role of transcranial Doppler: A systemic review of the literature. Cardiovasc Ultrasound 2006; 4: 47.

29- Alvarez-Fernandez JA, Martin-Velasco MM, Igeno-Cano JC , Perez-Quintero R. Transcranial Doppler ultrasonography usefulness in cardiac arrest resuscitation. Med Intensiva 2010;34(8): 550-8.
30- Edgren E, Enblad P, Grenvik A, Lilj A, Valind S, et al. Cerebral blood flow and metabolism after cardiopulmonary resuscitation: A pathophysiologic and prognostic positron emission tomography a pilot study. Resuscitation 2003; 57(2): 161-70.
31- Leonov Y, Strez F, Safar P, Johnson DW, Tisherman SA, et al. Hypertension with hemodilution prevents multifocal cerebral hypoperfusion after cardiac arrest in dogs. Stroke 1992; 23(1): 45-53.
32- Kochanek PM and Hallenbeck JM. Polymorphonuclear leukocytes and monocytes/macrophages in the pathogenesis of cerebral ischemia and stroke. Stroke 1992; 23(9): 1367-79.
33- Krep H, Brinker G, Schwindt W, Hossmann KA. Endothelin type A- antagonist improves long-term neurological recovery after cardiac arrest in rats. Crit Care Med 2000; 28(8): 2873-80.
34- Bunk G, Van der Hoeven JG, Frolich M, Meinders AE. Cerebral vasoconstriction in comatose patients resuscitated from cardiac arrest? Intensive Care Med 1996; 22(11): 1191-6

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مدى صحة الاستخدام المتلازم الغير اختراقي بجانب السرير للفحص الإكلينيكي وجهاز الموجات الصوتية دوبلر عبر الجمجمة للتوقع بالنتاج بعد توقف القلب

المشاركون في البحث
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ان تقييم التشافي العصبي بعد عمليه الإنعاش الناجحة للمرضي الذين اصيبوا بتوقف القلب قد يتم بالفحص الاكلينيكي . وكان الهدف من هذه الدراسه تقييم تلازم استخدام جهاز الموجات الصوتيه دوبلر عبر الجمجمة بالاضافه الي الفحص الاكلينيكي لتوقع النتاج بعد عمليه الانعاش القلب رئويه . و قد تمت هذه الدراسه علي عدد ٤٥ حاله توقف قلب اجريت لهم عمليات انعاش ناجحه ، تم الفحص الإكلينيكي للمرضي مشتملا (حرز جلاسجو للغيبوبه – الحرز الحركي ، منعكس الحدقه ، منعكس القرنيه) . اجريت هذه الفحوص في الفترات التاليه : بعد عمليه الانعاش ثم بعد ٢٤ ساعه ، بعد ٤٨ ساعه ، وبعد ٧٢ ساعه . ايضا تم قياس متوسط سرعه سريان الدم المخي و المنسب المتبثر باستخدام جهاز الموجات الصوتيه دوبلر عبر الجمجمة في نفس الاوقات السابق ذكرها . و بعد متابعه المرضي لمده ٢٨ يوما تم تقسيمهم الي مجموعتين طبقا لحرز الكفاءه المخيه: المجموعه الاولي (حرز الكفاءة المخيه ١ -٢) ذات نتاج جيد و المجموعه الثانيه (حرز الكفاءة المخيه ٣-٥) ذات نتاج ضعيف المستوي . و جاءت نتائج البحث كالأتي : نسبه ٢٨.٩ % من المرضي (ذو حرز الكفاءة المخيه ١ – ٢) كان نتاجهم جيد بينما نسبه ٧١.١% من المرضي (ذو حرز الكفاءة المخيه ٣-٥) كان نتاجهم ضعيف المستوي .و قد لوحظ ان عدد المرضي ذوي حرز جلاسجو للغيبوبه – الحرز الحركي اقل من او يساوي ٣ او من كان لديهم فقدان لمنعكس حدقه العين او منعكس القرنيه قد انخفض تدريجا علي فتره ٧٢ ساعه التاليه لعمليه الانعاش في المجموعه الاولي عن المجموعه الثانيه . هذا و قد كان متوسط سرعه سريان الدم المخي بعد عمليه الانعاش منخفضا في كلا المجموعتين ثم بعد ذلك كان هناك زياده تدريجيه بصوره ملحوظه احصائيا في فتره ٧٢ ساعه التاليه في المجموعه الاولي عن المجموعه الثانيه . ايضا كان متوسط قيم المنسب المتبثر مرتفعا في كلا المجموعتين بعد عمليه الانعاش و لكن كان هناك انخفاض ملحوظ احصائيا في هذه القيم في المجموعه الاولي عن المجموعه الثانيه خلال فتره ٧٢ ساعه التاليه. اثناء متابعه الحالات بعد مرور ٧٢ ساعه على عمليه الانعاش كان الفحص الاكلينيكي (حرز جلاسجو للغيبوبه – الحرز الحركي اقل من او يساوي ٣ ، و ايضا فقدان منعكس حدقه العين و القرنيه) ايجابي للتوقع بالنتاج ضعيف المستوي بنسبه ٩٢.٦ % و كانت الحساسيه ٧٨.١ % و الخصوصيه ٨٤.٦ % و الدقه ٨٠ % . كما كانت قياسات جهاز الموجات الصوتيه دوبلر عبر الجمجمة بعد مرور ٧٢ ساعه على عمليه الانعاش ايجابيه التوقع للنتاج ضعيف المستوي بنسبه ٩٦.٤% حيث كانت نسبه الحساسيه ٨٤.٤ % و الخصوصيه ٩٢.٣ % و الدقه ٨٦.٧ % . في حين كان تلازم استخدام الفحص الاكلينيكي و جهاز الموجات الصوتيه دوبلر عبر الجمجمة نتج عنه ارتفاع نسبه التوقع الايجابي للنتاج ضعيف المستوي الي ١٠٠ % و كانت الحساسيه ٩٠.٦ % و الخصوصيه ١٠٠ % و الدقه ٩٣.٣ % . و خلص هذا البحث الي اهميه تلازم استخدام الفحص الاكلينيكي و جهاز الموجات الصوتيه دوبلر عبر الجمجمة بعد مرور ٧٢ ساعة علي عملية الانعاش للتوقع الاكثر دقة بالنتاج بعد توقف القلب بدلا من استخدام طريقه واحده بمفردها.