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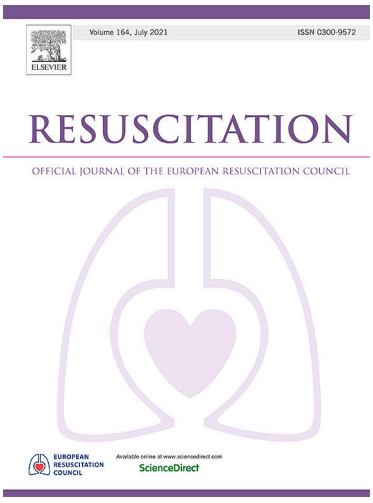

Letter to the Editor

Introducing the C's in CPR

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Introducing the C's in CPR

To the editor,

Identification and correction of cardiac arrest aetiology is of utmost importance in advanced life support (ALS). The patient's story preceding the arrest is important in differentiating between these aetiologies. Unfortunately this information is not always readily available, leaving the ALS team to consider the 'reversible causes' as published in the latest European Guideline on Advanced Life Support.[1] These are commonly referred to as the "4H's and 4T's".

A practical list of aetiologies to consider in cardiac arrest cannot include all conceivable causes. Therefore a balance is needed between concise for easy remembering and all-encompassing for optimised individual care. When the list becomes too concise, this might pose two specific problems:

First of all, aetiologies with distinct therapeutic implications are missed. Of particular relevance are aetiologies that might require adaptations of the standard advanced life support algorithm, e.g. hypertrophic cardiomyopathy and/or subarachnoid haemorrhage.

Secondly, erroneously fixation on a particular aetiology (and subsequent therapy) happens more easily.

The authors feel that an extension of the well-known acronym with the 5 C's of CPR results in a more concise, yet still easy to remember acronym that might benefit a wider population of cardiac arrest patients (see table 1).

Cerebral causes especially acute subarachnoid haemorrhage is known for its presentation with serious ECG abnormalities, including third degree AV block, ventricular tachycardia, ventricular fibrillation, and asystole.[2] This aetiology is especially relevant to consider regarding conflicting targets on (anti-)coagulation.

Cardiomyopathy (e.g. infectious, dilated, hypertrophic) might cause severe hemodynamic compromise and/or sudden cardiac death (SCD). The latter being often the first clinical presentation in children and young adults with hypertrophic cardiomyopathy.[3] Depending on the type of cardiomyopathy and aetiology of the arrest, additional therapeutic options might be applicable to the ALS algorithm.

Conduction abnormalities (e.g. Long- / Short-QT-syndrome, Brugada syndrome) are a well-known cause for sudden cardiac death.[1] Some of these might result secondary to causes within the original "4H's and 4T's" however this does not cover all of them, necessitating an explicit evaluation for conduction abnormalities.

Congenital abnormalities (e.g. structural heart disease, valvular heart diseases, coronary artery anomalies) are most likely diagnosed early in life. Often anatomic corrections of the

heart have been performed however this might not have happened (yet) and/or emergency care providers might not always be aware of previous interventions.

Commotio cordis is a well-described risk factor for SCD.[4] Although exact numbers are lacking due to the absence of systematic reporting, based on their local data Maron and Estes (2010) found commotio cordis amongst the most frequent causes in SCD in young athletes, after hypertrophic cardiomyopathy and congenital coronary artery anomalies,[4] i.e. two other proposed C's.

Hypoglycaemia is not included as an aetiology on itself. One might argue that it is included within the 'metabolic derangements' as mentioned with the 'H' of 'hypo- / hyperkalaemia'. This might however lead to a delay or even complete omission to consider glucose as a causative aetiology.[5]

Table 1: proposed acronym for identification cardiopulmonary arrest aetiology

Hypoxia	**T**hrombosis – coronary	**C**erebral cause (i.e. SAH)
Hypovolaemia	**T**hrombosis – pulmonary	**C**ardiomyopathy
Hypo-/hyperkalemia/metabolic	**T**ension pneumothorax	**C**onduction abnormality
Hypo-/hyperthermia	**T**amponade – cardiac	**C**ongenital
Hypoglycemia	**T**oxins	**C**ommotio cordis (i.e. traumatic)

SAH = subarachnoid haemorrhage

Conflict of interest

None

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