External cardiac compression may be harmful in some scenarios of pulseless electrical activity.

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SUMMARY

Pulseless electrical activity occurs when organised or semi-organised electrical activity of the heart persists but the product of the systemic vascular resistance and the increase in systemic arterial flow rate generated by the ejection of each left ventricular stroke volume is not sufficient to produce a clinically detectable pulse. Pulseless electrical activity encompasses a very heterogeneous ~~a~~ range of severe circulatory shock states ranging in severity from pseudo-cardiac arrest with significant persistant cardiac output to effective cardiac arrest with zero or negative cardiac output. Outcomes of cardiopulmonary resuscitation for pulseless electrical activity are generally poor. Impairment of cardiac filling is the limiting factor to cardiac output in many scenarios of pulseless electrical activity, including extreme vasodilatory shock states. There is no evidence that external cardiac compression can increase cardiac output when impaired cardiac filling is the limiting factor to cardiac output. Repeated cardiac compression asynchronous with the patient's cardiac cycle and raised mean intrathoracic pressure due to chest compression can both be expected to reduce rather than to increase cardiac filling and therefore to reduce rather than to increase cardiac output in such circumstances. The hypothesis is proposed that the performance of external cardiac compression will have zero or negative effect on cardiac output in pulseless electrical activity when impaired cardiac filling is the limiting factor to cardiac output. The performance of external cardiac compression may be both directly and indirectly harmful to significant sub-groups of patients with pulseless electrical activity. If this is the case, then chest compression-only cardiopulmonary resuscitationcan be predicted to be particularly harmful. We have neither evidence nor theory to provide comfort that external cardiac compression is not harmful in many scenarios of pulseless electrical activity. Investigation using a variety of animal models of pulseless electrical activity produced by different shock-inducing mechanisms is required to provide an evidence base for guidelines.

INTRODUCTION

The term *pulseless electrical activity* (PEA) refers to persistent organised or semi-organised electrical activity of the heart in patients who lack a detectable pulse.

PEA occurs in a very heterogeneous group of underlying conditions and can be produced by a variety of very different circulatory shock-inducing mechanisms. These mechanisms are impaired cardiac filling, impaired cardiac pumping, loss of systemic vascular resistance (SVR) and circulatory obstruction, most commonly due to acute pulmonary embolism. More than one mechanism may be involved in any given case. PEA occurs when organised or semi-organised electrical activity persists but the product of the SVRand the increase systemic arterial flow generated by the ejection of each left ventricular stroke volume is not sufficient to produce a clinically detectable pulse.

The diagnosis of pulselessness may be affected by many variable factors including the level of cardiac output, the systemic vascular resistance, the skill of the observer, environmental factors, and patient factors such as reduced body temperature or obesity. Because of these variables, it can be expected that patients will be deemed to be pulseless at very different cardiac output levels from case to case.

PEA is currently categorised as a form of cardiac arrest. However, prompt and effective treatment of identified conditions causing PEA, such as tension pneumothorax, cardiac tamponade, hypovolaemia and vasodilatory states, can produce a return of a detectable pulse without any cardiac treatment. Such haemodynamic improvement resulting in the return of a detectible pulse clearly does not equate to the re-starting of a stopped heart.

Significant cardiac output has been demonstrated in some patients with clinical diagnoses of PEA. (1, 2) These patients have been categorised as having “pseudo-PEA” and the term pseudo-mechanico-electrical dissociation has previously been accepted for this scenario.(1,3) The validity of these

terms is highly questionable. While the diagnosis of pulselessness in these cases may be misleading it is not accurately described as pseudo-puselessness, nor is the electrical activity of the heart correctly described as pseudo-organised electrical activity. PEA where significant cardiac output persists is surely more correctly described as *pseudo-cardiac arrest* than as *pseudo-PEA*. The acceptance of a diagnostic category of pseudo-PEA would require that a diagnosis of (true) PEA could only be made after the use of technology to out-rule significant persistent cardiac output. In other words the diagnosis of PEA could not be made based on clinical failure to detect a pulse. This would require a redefinition of PEA. Using the current definition of PEA, it is more correct to say that PEA encompasses a very heterogeneous ~~a~~ range of severe circulatory shock states ranging in severity from pseudo-cardiac arrest through near-cardiac arrest to effective cardiac arrest.

Outcomes of CPR for the treatment of PEA are generally very poor. For example, in the SOS-KANTO study only 2% of patients who received standard CPR by bystander for out-of hospital PEA had a good neurological outcome at thirty days.(4) However, PEA does not intrinsically carry a poor prognosis because prompt therapeutic measures targeting identified underlying causes of pulselessness, as previously outlined, can produce good outcomes.

In view of the low success rate of CPR for PEA, and in view of the heterogeneous causative mechanisms of PEA, it is appropriate to consider the possibility that basic CPR may actually be harmful in some subgroups of PEA cases. Further, given that PEA includes a subset of patients in whom there is significant residual cardiac output, it is at least possible that the performance of ECC will impair rather than augment existing cardiac output in some scenarios of PEA. The performance of ECC in scenarios where it cannot increase cardiac output could also indirectly impair survival by delaying or by distracting from other potentially more useful actions aimed at diagnosing and treating the underlying cause of PEA or aimed at attenuating the relevant shock-inducing mechanisms. This indirect negative therapeutic effect could occur even in the absence of significant cardiac output.

BACKGROUND EVIDENCE

There are no published data on the effect of ECC on cardiac output or systemic perfusion across the spectrum of heterogeneous scenarios of PEA. There are also no outcome studies which provide evidence relating to the comparative value of ECC in PEA produced by different causative mechanisms.

There is evidence suggesting that vigorous ECC is of value in PEA due to pump failure and due to circulatory obstruction as a consequence of pulmonary embolism. (5) These scenarios of PEA have in common the fact that the heart is not capable of pumping out all of the blood returning to it. No such evidence is available for PEA due to reduced cardiac filing or for PEA due to reduced SVR and associated increased systemic vascular capacitance. These scenarios have in common that the heart is, at least in the early stages of PEA, capable of pumping out all of the blood returning to it and that reduced cardiac filling, rather than impaired ejection, is the cardiac output-limiting factor.

DISCUSSION

In the absence of evidence supporting the performance of ECC in PEA due to reduced cardiac filing or reduced systemic vascular resistance, and in the light of the current poor outcomes of CPR, it is worth considering the possible effects of repeated chest and cardiac compression in these scenarios.

Impaired cardiac filling

Scenarios of PEA in which inadequate cardiac filling is the primary causative factor include haemorrhagic hypovolaemia, tension pneumothorax, and cardiac tamponade. Impaired cardiac filling, due to relative hypovolaemia as a consequence of increased systemic vascular capacitance, may also be the limiting factor to cardiac output in scenarios of PEA due to vasodilatory states.

If impaired cardiac filling is the limiting factor to cardiac output and the heart is effectively ejecting all the blood returning to it, then ECC can only increase cardiac output if it increases venous return and cardiac filling.

The pressure gradient underlying venous return from the systemic circulation to the right atrium is normally low and it is well recognised that modest increases in mean intra-thoracic pressure can impair venous return, particularly when venous return is already challenged or compromised, typically in hypovolaemic patients who have poor tolerance of raised intra-thoracic pressure due to positive pressure ventilation.

50% or more of the cardiac compressions of ECC in CPR for PEA can be expected to occur during diastole. Repeated compression of the heart in diastole can be expected to impair cardiac filling. The increase in mean intra-thoracic pressure produced by ECC can also be predicted to impair venous return and cardiac filling. If impaired cardiac filling is the prior limiting factor to cardiac output, then performance of ECC, *prima facia,* will impair rather than augment cardiac output. If cardiac output is significant, its reduction must reduce the patient's chances of survival. If cardiac output is insignificant as a result of impaired cardiac filling, then clearly ECC cannot significantly impair output but it also appears to be highly unlikely that ECC in this circumstance can significantly increase cardiac output. The chances of survival could also be impaired indirectly by the distraction of performing ECC rather than concentrating on potentially more productive interventions aimed at improving cardiac filling.

From this simple theoretical analysis it appears that in cases of PEA due to impaired cardiac filling the performance of ECC will, at best, be therapeutically useless and that it will possibly be detrimental to survival. There appears to be no evidence to contradict this view which is reflected in the aphorism that "there is no point in massaging the empty heart".

It appears entirely reasonable to propose that the performance of ECC will be detrimental to survival and to neurological outcome when PEA is due to impaired cardiac filling.

Reduced Systemic Vascular Resistance (Vasodilatory States)

Systemic perfusion pressure is a simple product of cardiac output and SVR. Large falls in SVR can occur physiologically in vigorous exercise and in various pathological and iatrogenic scenarios. SVR can be estimated to be less than 0.26 of normal resting levels in a non-extreme physiological scenario of vigorous exercise based on published data on cardiac output and systemic arterial blood pressure in exercise. (6) Similar calculation for more extreme exercise scenarios of greater than 20 metabolic unit equivalents (MET) estimate SVR at less than 0.2 of normal resting levels.

Vasodilatory states with reduced SVR are the cause of pulselessness in several PEA scenarios including anaphylaxis, acute high spinal cord injuries, accidental total spinal anaesthesia, accidental vasodilator overdose and accidental cessation of inotropic/vasopressor infusions in severe vasodilatory shock states such as septic shock. Reduced SVR may also be a significant contributing factor in post-defibrillation PEA after acute shockable rhythm cardiac arrest of onset in vigorous exercise and in other scenarios of pre arrest reduction in SVR. Reduced SVR will generally be associated with increased systemic vascular capacitance and therefore with reduced cardiac filling due to relative hypovolaemia resulting and reduced central venous pressure. As previously suggested, ECC can reasonably be predicted to impair cardiac output in such circumstances. (6)

Patients in whom loss of SVR is the primary cause of PEA may have significant cardiac output which is undetected due to low pulse pressure as a consequence of low SVR. For example, if SVR is 0.25 of normal, a cardiac output of 0.6 of normal (3 Litres $\cdot$ min^{-1} in the Textbook Man) will produce a mean systemic perfusion pressure of only 0.15 of normal resting level or approximately 13.5 mmHg in the textbook case. Pulse pressure will be extremely low in such circumstances and no pulse will be detectable resulting in a diagnosis of PEA “cardiac arrest” in spite of the presence of substantial

persistent cardiac output. If, as a consequence of associated increased vascular capacitance, impaired cardiac filling is a limiting factor to cardiac output then the potential arises that ECC can cause significant reduction in cardiac filling and in cardiac output in such a scenario. The mechanisms again being impairment of cardiac filling due to raised mean intrathoracic pressure and by asynchronous cardiac compression with many compressions occurring during diastole

It is a further consideration that with a significant residual cardiac output ventilation of the lungs will be of greater importance for resuscitation due to significant pulmonary oxygen uptake during any pre-resuscitation interval and during resuscitation.

Poor outcomes have been reported for cardiac arrest in of onset during vigorous exercise in college athletes, in spite of prompt expert resuscitation efforts. (7) Post defibrillation PEA was identified as a particular feature in this report. As has previously been suggested, the performance of ECC in post defibrillation PEA in this scenario may well be contributing negatively to survival in some of these patients with greatly reduced SVR and increased vascular capacitance due to large-scale metabolically-linked sympatholytic vasodilatation in skeletal muscles.(6)

Conclusion

When PEA ~~which~~ is due to impaired cardiac filling, with or without loss of SVR, and where the heart is already effectively pumping out the blood which is returning to it, it appears to be likely, or at least possible, that ECC cannot increase cardiac output and that it may decrease undetected residual cardiac output.

HYPOTHESIS PROPOSAL

It is proposed that consideration of the available evidence and of the underlying pathophysiology of PEA allows the proposal of the following hypothesis.

H**ypothesis**

External cardiac compression will have zero or negative effect on cardiac output in pulseless electrical activity when cardiac filling is the limiting factor to cardiac output.

CURRENT EVIDENCE

There is no evidence currently available which either directly supports or refutes this hypothesis.

POTENTIAL IMPLICATIONS

If the hypothesis is true then ECC must be harmful to some patients with PEA and may be contributing to the current poor outcomes of CPR for PEA. It must also be considered that if ECC is harmful to some patients with PEA, it is likely that chest compression-only resuscitation will be particularly harmful in these cases.

Even without proof of the proposed hypothesis, the current unstated assumption that ECC is of positive or zero benefit in all cases of PEA is not supported by evidence, therefore we do not currently have adequate evidence to support the immediate commencement of ECC in all scenarios of PEA. A more sophisticated balance of risk approach, depending on the clinical scenario, may be required to the performance ECC in PEA. This could be supported by end-tidal carbon dioxide monitoring which could provide evidence of the persistence of significant spontaneous cardiac output and evidence as to whether ECC, if commenced, results in increased or decreased cardiac output or in no change.

TESTING OF THE HYPOTHESIS

Testing of the hypothesis requires assessment of the effect ECC on cardiac output and systemic perfusion pressure in a variety of animal models of PEA produced by the different shock-inducing mechanisms. The results of resuscitation with ECC as compared to resuscitation without ECC could also be assessed in such models.

CONCLUSION

PEA is arguably more correctly described as an extreme circulatory shock state than as a state of cardiac arrest.

ECC could have negative therapeutic utility in scenarios of PEA where impaired cardiac filling and loss of SVR are the primary causative mechanisms.

The performance of ECC in PEA due to reduced cardiac filling and to vasodilatory states could be contributing both directly and indirectly to the poor outcomes of CPR for PEA. If this is the case, then chest compression-only CPR is likely to be particularly harmful. This will be particularly true in cases of pseudo-cardiac arrest where there will be greater requirement for ventilation of the lungs to prevent development of systemic arterial hypoxaemia.

A more circumspect balance of risk approach is required, particularly by physician resuscitators, to the performance ECC in PEA. This could be supported by the use of end-tidal carbon dioxide monitoring.

Investigation using a variety of animal models of PEA produced by the different shock-inducing mechanisms is required to provide an evidence base for guidelines.

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