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Research Article

A Computer-based Study on the Effect of Sympathetic Activity during CPR

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ARTICLE INFO

Article history:
Received 22 November, 2018
Accepted 17 December, 2018
Published 23 December, 2018

Keywords:
Cardiac arrest
cardiopulmonary resuscitation
cardiac output
mean arterial pressure
computer model

ABSTRACT

Purpose: In spite of extensive studies, the mechanism of cardiopulmonary resuscitation (CPR) has not been properly understood and a proper comprehension of the role of regulatory mechanisms of the cardiovascular system during CPR is unavailable. Using computational methods, we try to study the influence of sympathetic activation on the cardiac output and mean arterial pressure (MAP) during CPR at different compression pressures and rates.

Methods: A computer model was used to investigate the effect of sympathetic activation during CPR. The model has a detailed representation of the cardiopulmonary resuscitation system and sympathetic control. Sympathetic activation during CPR was achieved through vital cardiac parameters such as contractility and peripheral resistance. We compared the cardiac output and MAP during CPR in four scenarios, namely with; (1) sympathetic activation of heart alone, (2) sympathetic activation of the peripheral arteries alone, (3) sympathetic activation of both heart and peripheral arteries, and (4) no sympathetic activation; for different compression pressures and rates.

Results: The results show that the cardiac output and MAP increases with increasing compression pressures and rates during CPR with sympathetic activation of peripheral arteries. The sympathetic activation of peripheral arteries during CPR at the AHA and ERC recommended chest compression pressures and rates resulted in an increased MAP, an augmented aortic diastolic pressure and a decreased cardiac output. The results also show that cardiac output and MAP pressure increases with increasing compression rate during CPR with sympathetic activation of heart. There is a slight increase in the MAP but no substantial improvement in cardiac output during CPR with sympathetic activation of heart at the AHA and ERC recommended pressures and rates.

Conclusions: It is observed from the study that sympathetic activation of heart during CPR may not be beneficial at the AHA and ERC recommended chest compression rates as it gives very little improvement in cardiac output and MAP. However, performing CPR at higher compression rate may improve the chances of resuscitation when drugs are used to induce sympathetic activity in the heart. The augmented aortic diastolic pressure during CPR with sympathetic activation of peripheral arteries at the AHA and ERC recommended compression pressure and rates can improve the myocardial perfusion, but the reduced cardiac output is a cause of concern.

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http://dx.doi.org/10.31487/j.JICOA.2018.01.004

Introduction

Cardiopulmonary resuscitation (CPR) is an emergency lifesaving procedure that helps maintain life in a person whose heart has stopped beating. Even with widespread usage and extensive studies, the mechanism of blood flow during CPR continues to be uncertain and poorly understood. Nishizawa et al. and Sundgreen et al. found the auto-regulation impaired in patients resuscitated from cardiac arrest, but a proper comprehension of the role of regulatory mechanism of the cardiovascular system during CPR is still unavailable [1, 2].

A number of animal models have been used to which study the effect of sympathetic activity during CPR. Otto and Yakaitis studied the use of drugs during cardiac arrest in dogs and observed that the drugs used to achieve sympathetic activation of heart during CPR have no value in the therapy of cardiac arrest [3]. Redding and Pearson studied CPR in dogs and observed that while drugs inducing sympathetic activation of heart are not useful for resuscitation, the drugs inducing sympathetic activation of peripheral arteries are useful [4]. Yakaitis et al. found that sympathetic activation of heart in dogs has little effect on successful resuscitation but the sympathetic activation of peripheral arteries during CPR improves the diastolic pressure, which is important for successful resuscitation [5]. Livesay et al. also studied the effect of drugs which cause the sympathetic activation of peripheral arteries during CPR in dogs and noted that the sympathetic activation of peripheral arteries results in an augmented aortic diastolic pressure, improving the coronary perfusion during CPR [6]. The work of Pearson and Redding worked on a dog model to show that drugs which cause a sympathetic activation of peripheral arteries during CPR improves the diastolic pressure, thereby improving the chances of spontaneous circulation [7]. The findings of Gonzalez et al. show that the use of drugs for sympathetic activation peripheral arteries in human beings also results in an increased aortic diastolic pressure during CPR [8]. The study of Micheal et al. also on dogs, noted that the drugs inducing sympathetic activation of heart improves the myocardial blood flow during CPR [9].

Even though a number of animal models studied the effect of sympathetic activation during CPR, none of these models studied the influence of sympathetic activity on the cardiac output and MAP at different compression pressures and rates. We used a computer modelling approach to study the effect of sympathetic activation during CPR at different compression pressures and rates. Using a computer model gave us the flexibility to perform experiments that are otherwise difficult to be performed on human subjects or animals.

Our model has a detailed representation of cardiopulmonary resuscitation system and sympathetic control. The sympathetic activity on the cardiovascular system during CPR was achieved through heart contractility and peripheral resistance.

Materials and Methods

I Cardiopulmonary Resuscitation (CPR) Model: Our earlier Cardiopulmonary Resuscitation (CPR) model reported in John et al. was used to study the effect of sympathetic activation during CPR as it can simulate the CPR physiology under various conditions [10]. This CPR model, as shown in (Figure 1), is complete with veins, vena cava, right atrium, tricuspid valve, right ventricle, pulmonary valve, pulmonary artery, pulmonary capillary, pulmonary veins, left atrium, mitral valve, left ventricle, aorta, arteries, and capillaries. This is a lumped element model consisting of resistors, capacitors and inductors, and is represented using 27 simultaneous differential equations, which are solved using 4th order Runge-Kutta method.

The model consists of four cardiac chambers and each cardiac chamber wall property is modelled with two elements – elastance and viscoelastic resistance, to represent internal resistance of the chamber. Elastance of both the ventricles are assumed to be a constant in this CPR model. The heart valves are considered as orifices and since pressure-flow relationship across any orifice is defined by Bernoulli's law, the valves are modelled using Bernoulli's resistance, inertance and resistance. The initial volume in each chamber is adjusted to set the pressure of the chamber at 15 mmHg, which is the initial filling pressure. In this CPR model, the minimum volume in each element is limited to the residual volume. The residual volume values for each element is from the work by Koeken et al. and the nominal value for each element in our CPR model are given in Appendix I [11].

The chest compression pressure in this CPR model is given as intrathoracic pressure. The compression pressure is applied equally to all thoracic elements. CPR is simulated in the model using compression pressure as input, which are sinusoidal pulses to mimic manual CPR. The duration where the pressure builds up is the compression period and the remainder of the cycle is the relaxation period.

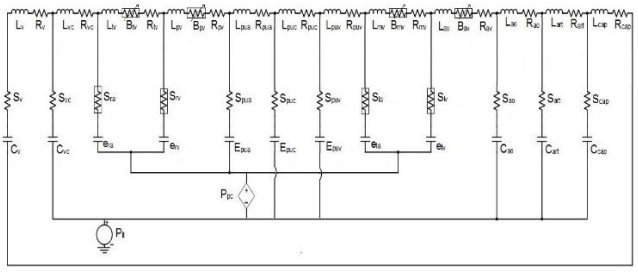

Figure 1: The electrical equivalent of CPR model. S, C, E, L, B and R-viscoelastic resistance, capacitance, elastance, inertance, Bernoulli's resistance, and resistance respectively. P_{pc} - Pericardium pressure. P_{it} – intrathoracic pressure. Suffix explanation: v - venous, vc - vena cava, ra - right atrium, tv - tricuspid valve, rv - right ventricle, pv - pulmonary valve, pua - pulmonary artery, puc - pulmonary capillary, puv - pulmonary vein, la - left atrium, mv - mitral valve, lv - left ventricle, av - aortic valve, ao - aorta, art - artery, and cap - capillary.

II Sympathetic System: Sympathetic activity plays an important role in the pressure regulation by adjusting heart rate, cardiac contractility and peripheral vascular resistance in a healthy human being. A detailed description of the sympathetic system was therefore incorporated in the present model to study its effect during CPR. The sympathetic system was developed based on Ursino's model [12]. The effect of sympathetic activation was coupled with left ventricular elastance, right ventricular elastance, and peripheral resistance.

Aortic pressure was the input to aortic baroreceptors and firing frequency was the output. The afferent pathway function was achieved by a first-order state equation and a sigmoidal static function. The mean point of the sigmoidal function was assumed to be 96 mmHg, which is

approximately equal to the mean aortic pressure in a healthy human. The efferent reflexes are mediated by sympathetic nerves and its firing frequency is inversely proportional to the baroreceptor stretching rate. Therefore, the sympathetic efferent activity was realised by a monotonically decreasing exponential curve. Each sympathetic efferent pathway was modelled by a generic monotonic logarithmic function with pure delay. The mathematical equations and parameters were same as in the Ursino's model [12].

Our model was developed in MATLAB R2010a with a fixed step-size of 0.001s, which was of the order of the smallest time constant of the system.

We studied the cardiac output and MAP during CPR in the following 4 scenarios:

1. with sympathetic activation of heart alone (SH),
2. with sympathetic activation of peripheral arteries alone (SP),
3. with sympathetic activation of both heart and peripheral arteries (SHP), and
4. with no sympathetic activation (NS), for a range of compression pressure and rate.

The results are showed as the mean cardiac output and mean arterial pressure over one minute after 30 seconds of compressions.

Results

I Cardiac Output Analysis

I. I Compression Pressure Analysis: The cardiac output during CPR with sympathetic activation of heart (SH), sympathetic activation of peripheral arteries (SP), sympathetic activation of both heart and peripheral arteries (SHP), and also without sympathetic activation (NS), were compared for different compression pressures in range of 50 mmHg to 150 mmHg at a constant compression rate of 110 compressions per minute (CPM). The cardiac output during CPR with SP and SHP was lesser than the cardiac output during CPR with no sympathetic activation of peripheral arteries for all compression pressures less than 130 CPM (as shown in figure 2). There was a 41.72% and a 38.08% decrease in cardiac output with sympathetic activation of peripheral arteries at 50 mmHg and 100 mmHg, respectively. However, there was a 43.59% increase in cardiac output with sympathetic activation of peripheral arteries at a compression pressure of 150 mmHg.

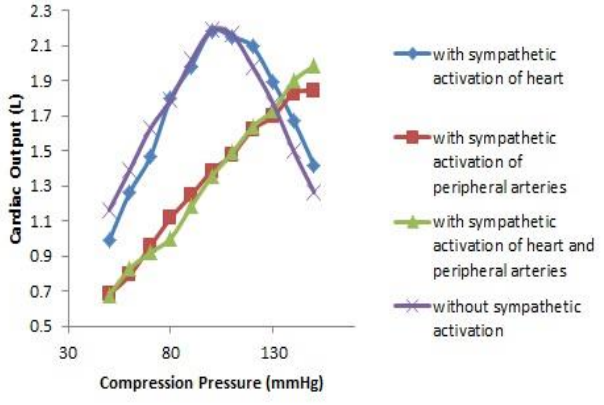

Figure 2: Pressure effects on cardiac output during CPR. Cardiac output vs. compression pressure at 110 CPM is shown.

As seen from figure 2, during CPR without any sympathetic activation of peripheral arteries, the maximal cardiac output is clearly distinguishable at a compression pressure of 100 mmHg. However, with sympathetic activation of peripheral arteries, the cardiac output increases with increasing compression pressure.

I. II Compression Rate Analysis:

The cardiac output during CPR with SH, SP, SHP, and NS were compared for different compression rates in the range of 60 CPM to 200 CPM at a constant compression pressure of 100 mmHg. The cardiac output during CPR with SHP and SP was lesser than that without any sympathetic activation of peripheral arteries for all ranges of compression rate as shown in fig. 3. The fall in cardiac output with sympathetic activation of peripheral arteries at 80 CPM, 110 CPM and 160 CPM were 37.15%, 40.05%, and 24.9%, respectively.

In figure 3, the maximal cardiac output is clearly distinguishable at a compression pressure of 100 mmHg in the CPR with NS. SH gives the largest cardiac output at higher compression rates. The cardiac output is seen to increase with increasing compression pressure in the CPR with SP, SH and SHP.

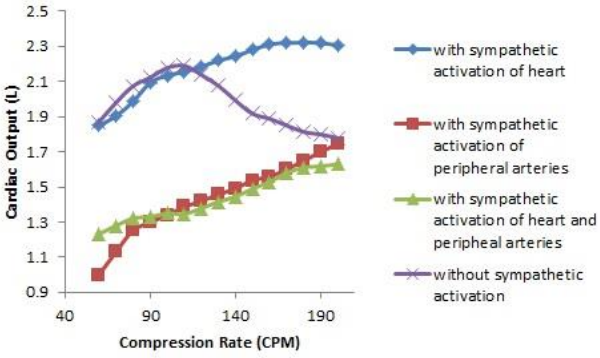

Figure 3: Rate effects on cardiac output during CPR. Cardiac output vs. compression rate at 100 CPM is shown.

II Mean Arterial Pressure (MAP) Analysis

II. I Compression Pressure Analysis: MAP during CPR with SH, SP, SHP and NS were compared for different compression pressures in range of 50 mmHg to 150 mmHg at a constant compression rate of 110 CPM. The MAP was greatest in the CPR with SHP and SP for all ranges of compression pressure, as shown in fig. 4. It is the sympathetic activation of peripheral arteries that gave an increased MAP. There was a 17.62% and an 81.05% increase in MAP with sympathetic activation of peripheral arteries at 50 mmHg and 150 mmHg, respectively. The aortic diastolic pressure increases only by 2.63% and 3.7% at 50 mmHg and 150 mmHg, respectively during CPR with sympathetic activation of heart. But the aortic diastolic pressure increases by 27.89 % and 75.06% at 50 mmHg and 150 mmHg during CPR with the sympathetic activation of peripheral arteries.

As shown in (Figure 4), the maximal mean arterial pressure is clearly distinguishable at a compression pressure of 100 mmHg in CPR without any sympathetic activation of peripheral arteries. However, in CPR with sympathetic activation of peripheral arteries, the MAP is seen to increase with increasing compression pressure.

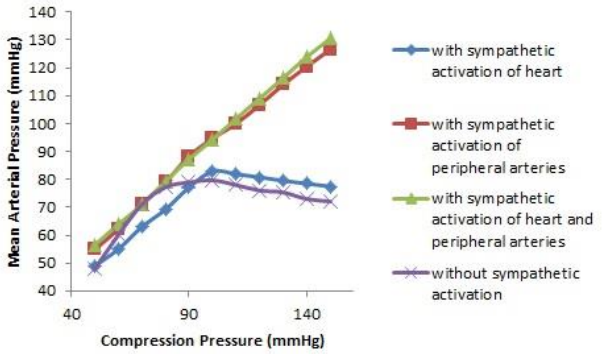

Figure 4: Pressure effects on mean arterial pressure (MAP) during CPR. MAP vs. compression pressure at 110 CPM is shown.

II. II Compression Rate Analysis: MAP with SH, SP, SHP and NS were compared for different compression rates in the range of 60 CPM to 200 CPM at a constant compression pressure of 100 mmHg. The MAP was greatest in the CPR with SHP and SP for all ranges of compression rates, as shown in fig. 5. The sympathetic activation of peripheral arteries gave this increased MAP. The MAP during CPR with sympathetic activation of peripheral arteries at 80 CPM, 110 CPM, and 160 CPM increased by 21.48%, 18.3%, and 35.01%, respectively. The aortic diastolic pressure increases by 2.91%, 1.51% and 10.11% at 80 CPM, 110 CPM and 160 CPM, respectively during CPR with sympathetic activation of heart. But the aortic diastolic pressure increases by 34.37 %, 27.28% and 28.11% at 80 CPM, 110 CPM and 160 CPM during CPR with the sympathetic activation of peripheral arteries.

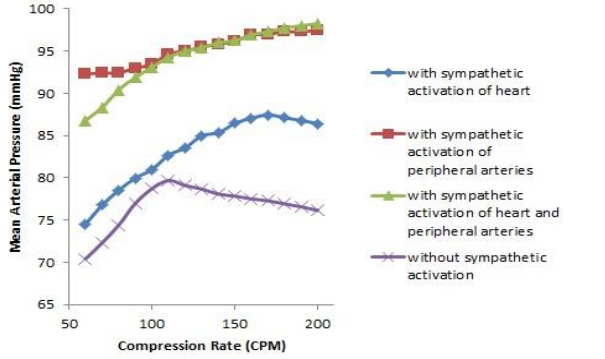

Figure 5: Rate effects on mean arterial pressure during CPR. MAP vs. compression rate for 100 mmHg is shown.

Without any sympathetic activation, the maximal MAP is at 110 CPM for 100 mmHg as shown in (Figure 5). However, with sympathetic activation the MAP is seen to increase with increasing compression rate.

Discussions

As per AHA 2015 and ERC 2015 guidelines for CPR, the recommended chest compression rate is 100 to 120 CPM and the recommended compression depth is approximately 6 cm [13, 14]. In our earlier work, we showed that the optimum compression depth for CPR is 5.7 cm, which corresponds to 100 mmHg of compression pressure [10].

Impact of Sympathetic activation on the cardiac output during CPR: Our results show that sympathetic activation of peripheral arteries during CPR at the AHA and ERC recommended chest compression pressures and rates results in a decreased cardiac output. It is because of the increased peripheral resistance from sympathetic activation of peripheral arteries that there is a reduction in the amount of blood flowing into the arteries. This reduced cardiac output during CPR with sympathetic activation of peripheral arteries can be detrimental to the effectiveness of CPR. Our results show that with the sympathetic activation of peripheral arteries during CPR, the cardiac output increases with increasing compression pressure since it avoids the collapse of vessels at higher compression pressures, giving an improved cardiac output. However, the cardiac output falls at higher compression pressures for CPR with no sympathetic activation of peripheral arteries since the flow gets obstructed due to the collapse of the vessels at higher compression pressures. We observe that with the sympathetic activation of peripheral arteries during CPR, the cardiac output also increases with increasing compression rate because of an increased preload. However, the cardiac output falls at higher compression rates for CPR with no sympathetic activation of peripheral arteries since the heart gets less and less time to get filled, reducing the preload.

Our simulation results also show that sympathetic activation of heart during CPR at the AHA and ERC recommended chest compression pressures and rates do not give an improvement in cardiac output. However, there is a substantial improvement in cardiac output at higher compression rates because of the increase in heart contractility during the CPR with sympathetic activation of heart.

Impact of Sympathetic activation on the MAP during CPR: The maximal MAP was with the sympathetic activation of peripheral arteries during CPR for all ranges of compression pressure and rate. This was expected as the sympathetic activation of peripheral arteries results in an increased blood pressure. In our model, the sympathetic activation of peripheral arteries during CPR also results in an augmented aortic diastolic pressure. Several studies on dogs and a study on human beings also show that the sympathetic activation of peripheral arteries improves the artificial aortic diastolic pressure [5 - 8]. The literature also notes that an improvement in the aortic diastolic pressure during CPR is of prime importance in augmenting the coronary perfusion pressure and that the myocardial perfusion improves the chances of return of spontaneous circulation [6, 7, 15].

We observe from our simulation results that sympathetic activation of heart during CPR at the AHA and ERC recommended chest compression rates do not give an increased MAP. Nevertheless, there is an improved MAP at higher compression rates during the CPR with sympathetic activation of heart.

According to our results, at the AHA and ERC recommended chest compression pressure and rates, the sympathetic activation of peripheral arteries results in an increased MAP but a diminished cardiac output, which can be detrimental. However, the cardiac output during the CPR with sympathetic activation of peripheral arteries shows an improvement at higher compression pressures and rates. Therefore, when drugs are used to induce sympathetic activation of the peripheral arteries during CPR, it might be beneficial to perform CPR at a compression pressure and rate higher than the AHA and ERC recommended value.

In our model, at the AHA and ERC recommended chest compression pressure and rates, the presence or absence of heart activation does not change the cardiac output or the MAP. These results are supported by

animal studies that show that the drugs which cause sympathetic activation of heart during cardiac arrest are of no value [3-5]. However, there is an improvement in cardiac output and MAP at higher compression rates during CPR with sympathetic activation of heart. Hence, if drugs are used to induce sympathetic activation of the heart during CPR, doing CPR at a compression rate higher than the AHA and ERC recommended value might be useful.

Conclusion

A computer model was developed to determine the effect of sympathetic activation on the hemodynamics during CPR. The CPR with sympathetic activation of peripheral arteries resulted in an increased aortic diastolic pressure and MAP, and a decreased cardiac output at the AHA and ERC recommended chest compression pressures and rates. However, since the cardiac output improves at higher compression rates and pressure during CPR with sympathetic activation of peripheral arteries, we conclude when using drugs to induce sympathetic activation of peripheral arteries, it might be useful to perform CPR at higher compression pressures and rates. The CPR with sympathetic activation of heart gives no improvement in cardiac output and MAP at the AHA and ERC recommended chest compression rates. Since the cardiac output and MAP increases with increasing compression rates, it might be useful to perform CPR at higher compression rates when drugs are used to achieve sympathetic activation of heart.

The current study used the sympathetic system model with effector values for a healthy human, even though there might have been a change in the effector sensitivity during cardiac arrest. The cerebral blood flow during this pathological condition was also not explored because the model does not separately consider the cerebral system in systemic circulation. Enhancing the current model by eliminating these deficiencies would improve the accuracy of sympathetic simulation during CPR.

Appendix I

Parameter	Values	Parameter	Values
B_{tv}	0.000016 mmHg.s^2.ml^{-2}	L_{art}	0.0001 mmHg.s^{2}ml^{-1}
B_{pv}	0.000025 mmHg.s^2.ml^{-2}	L_{cap}	0.0005 mmHg.s^{2}ml^{-1}
B_{mv}	0.000016 mmHg.s^2.ml^{-2}	R_v	0.07 mmHg.s^{2}ml^{-1}
B_{av}	0.000025 mmHg.s^2.ml^{-2}	R_{vc}	0.001 mmHg.s^{2}ml^{-1}
C_v	100 ml/mmHg	R_{tv}	0.005 mmHg.s^{2}ml^{-1}
C_{vc}	30 ml/mmHg	R_{pv}	0.005 mmHg.s^{2}ml^{-1}
C_{ao}	0.9 ml/mmHg	R_{pua}	0.04 mmHg.s^{2}ml^{-1}
C_{art}	0.3 ml/mmHg	R_{puc}	0.04 mmHg.s^{2}ml^{-1}
C_{cap}	0.006 ml/mmHg	R_{puv}	0.005 mmHg.s^{2}ml^{-1}
E_{pua}	0.02 mmHg/ml	R_{mv}	0.005 mmHg.s^{2}ml^{-1}
E_{puc}	0.02 mmHg/ml	R_{av}	0.005 mmHg.s^{2}ml^{-1}
E_{puv}	0.02 mmHg/ml	R_{ao}	0.03 mmHg.s^{2}ml^{-1}
e_{LA}	0.07 mmHg/ml	R_{art}	0.75 mmHg.s^{2}ml^{-1}
e_{LV}	2.87 mmHg/ml	R_{cap}	0.35 mmHg.s^{2}ml^{-1}
e_{RA}	0.055 mmHg/ml	S_v	0.01 mmHg.s^{2}ml^{-1}
e_{RV}	0.52 mmHg/ml	S_{vc}	0.01 mmHg.s^{2}ml^{-1}
L_v	0.0005 mmHg.s^{2}ml^{-1}	S_{RA}	0.0005 mmHg.s^{2}ml^{-1}
L_{vc}	0.0005 mmHg.s^{2}ml^{-1}	S_{RV}	0.0005 mmHg.s^{2}ml^{-1}
L_{tv}	0.0002 mmHg.s^{2}ml^{-1}	S_{pua}	0.01 mmHg.s^{2}ml^{-1}
L_{pv}	0.0005 mmHg.s^{2}ml^{-1}	S_{puc}	0.01 mmHg.s^{2}ml^{-1}
L_{pua}	0.0005 mmHg.s^{2}ml^{-1}	S_{puv}	0.01 mmHg.s^{2}ml^{-1}
L_{puc}	0.0005 mmHg.s^{2}ml^{-1}	S_{LA}	0.0005 mmHg.s^{2}ml^{-1}
L_{puv}	0.0005 mmHg.s^{2}ml^{-1}	S_{LV}	0.0005 mmHg.s^{2}ml^{-1}
L_{mv}	0.0002 mmHg.s^{2}ml^{-1}	S_{ao}	0.01 mmHg.s^{2}ml^{-1}
L_{av}	0.0005 mmHg.s^{2}ml^{-1}	S_{art}	0.01 mmHg.s^{2}ml^{-1}
L_{ao}	0.0005 mmHg.s^{2}ml^{-1}	S_{cap}	0.01 mmHg.s^{2}ml^{-1}

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