Effect of Continous Positive Airway Pressure On Cardiac Output In Normal Healthy Volunteers

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Summery

There are various techniques used for supportive ventilation, such as negative pressure ventilation, intermittent positive pressure ventilation (IPPV) and intermittent mandatory ventilation (IMV). They all provide effective ventilatory support but with the expense of a fall in cardiac output which in certain circumstances interferes with normal physiology. Continuous positive airway pressure (CPAP) (Fig.I), a method of respiratory support proposed by Barach[1], was introduced in clinical practice by Gregory[2] , and Civetta[3] in the treatment of some forms of acute respiratory failure (ARF). We studied the effect of CPAP on cardiac output in seven healthy volunteers, by applying a pressure of +5 cm and +10 cm, on two separate occasions. The cardiac output dropped with the application of CPAP of +5 cm and +10cm, but unlike other methods not to a statistically significant level (Table I). On the other hand cardiac output rose to statistically signficant level (P=.006 and P=.0095) after stopping CPAP on two separate occasions. These changes have also been noticed by other studies[4,5].

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Introduction

Continous positive airway pressure is widely used in the treatment of respiratory failure. It is gradually replacing positive end expiratory pressure (PEEP) espically in post- anaesthetic recovery state. In respiratory failure where it is difficult to wean the patient off the ventilator, CPAP has been found to be a useful method for patient needing ventilatory support.

Chronic obstructive airway disease is fairly common and the patients usually go into respiratory failure, ultimately reaching to the state of cor pulmonale. At this stage they certainly would benefit from ventilatory support but, once started on it, will become dependant on it and it will be difficult to wean them off the ventilator. In such circumstances CPAP can be helpful. But as mentioned earlier the positive pressure affects cardiac output. To see the effect of CPAP on cardiac output we conducted this study in seven healthy normal volunteers, by applying CPAP and measuring cardiac output non-invesively.

'Patients' and Method

Seven healthy volunteers, 4 male age 26-38 years and 3 female age 28-33 years, served as our subjects for the study. They were all healthy non-smoker, with no long standing medical problems. The protocol included two visits one week apart. Cardiac output was measured non-invasively by Bomed made in Germany. Continouse positive airway pressure was applied via Ambu CPAP system (Fig I) with closely applied face mask. The study was conducted in Respiratery department, Western Infirmary, Glasgow. All subjects were consented and the protocol was approved by the West of Scotland Ethical Committee, Western Infirmary, Glasgow.

After the initial screaning, each subject was monitored via Bomed to check the cardiac output, heart rate and blood pressure by appropriately applying the chest leads at certain positions. CPAP with +5 cm pressure was applied via closely applied mask for eight minutes. The cardiac output was measured every minute. At the end of eight minutes CPAP was discontinued and cardiac output was measured again.

The same procedure was repeated at the second visit but this time applying +10 cm pressure.

Result

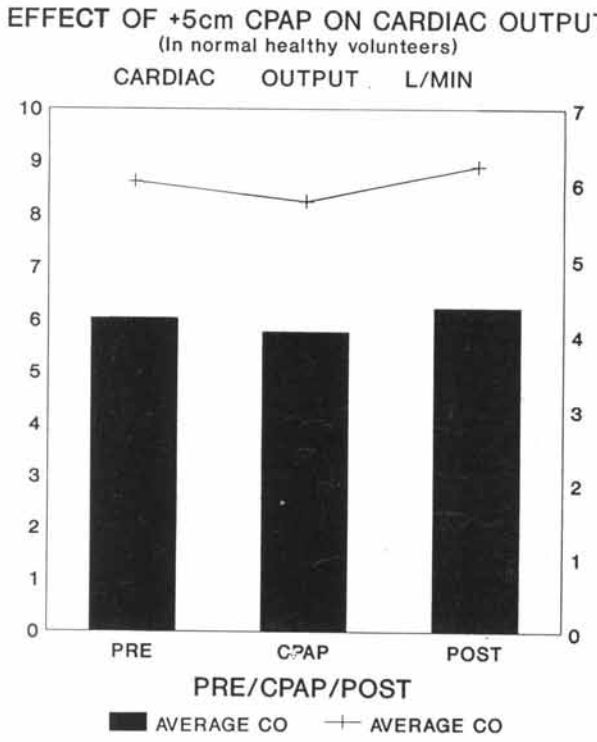
The average cardiac output bofeore, during and after CPAP of +5 cm, with standard error of the mean (SEM) were 6.03 (0.31), 5.77(0.21) and 6.23(0.29) respectively (Fig.2). There was a fall in cardiac output with CPAP but this fall was not statistically significant (P=0.15). But after stopping CPAP it rose to a statistically significant level (P=0.006).

Similarly the average cardiac output before, during and after CPAP of 10 cm, with SEM were 6.26 (0.26), 5.99(0.29) and 7.01(0.25) respectively (Fig.3). Again this fall in cardiac output was not statistically significant (P=0.11). This study suggests that CPAP does not effect the cardiac output and is farily tolerable.

TABLE-I

THE EFFECT OF +5cm & 10cm CPAP ON THE CARDIAC OUTPUT IN NORMAL HEALTHY VOLUNTEERS

Name	Pre CO c1	CO+5 c2	Post. c3	Pre CO c4	CO+10 c5	Post. c6
Iain M	5.4	5.2	5.6	6.7	6.2	7.6
Sandip G	6.2	6.4	6.6	7.1	7.3	7.4
Rita J	5.1	5.3	5.5	5.5	5.5	5.9
Paul R	5.8	5.6	6.1	5.5	5.3	7.6
Judith J	5.5	5.3	5.5	5.6	5.1	6.4
Jane M	6.8	6.0	6.8	6.5	6.5	7.3
Boeff H	7.4	6.6	7.5	6.9	6.0	6.9
Mean	6.03	5.77	6.23	6.26	5.99	7.01
(sem)	(0.31)	(0.21)	(0.29)	(0.26)	(0.29)	(0.25)



Pig.2 GRAPHIC REPRESENTATION OF THE AVERAGE EFFECT OF +5 cm CPAP ON CARDIAC OUTPUT IN NORMAL HEALTHY VOLUNTEERS.

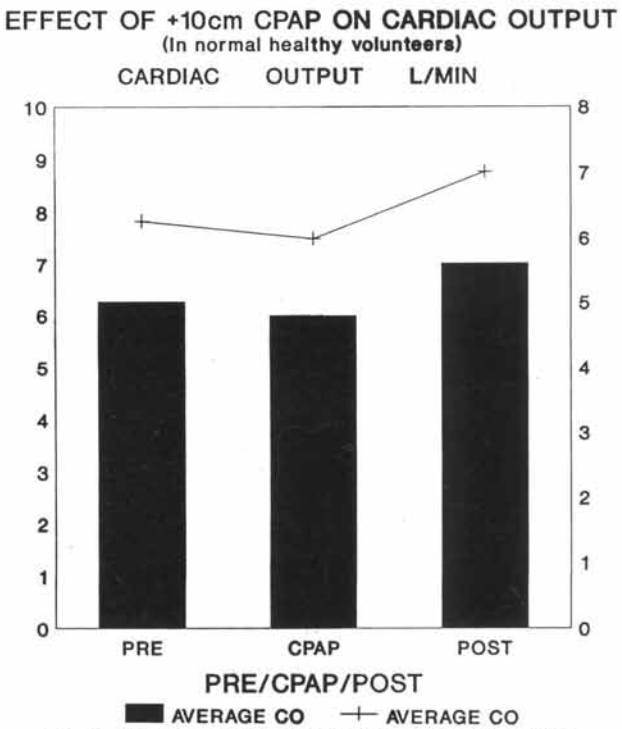


Fig.3 GRAPHIC REPRESENTATION OF THE AVERAGE EFFECTS OF + 10 cm CPAP ON CARDIAC OUT PUT IN NORMAL HEALTHY VOLUNTEERS.

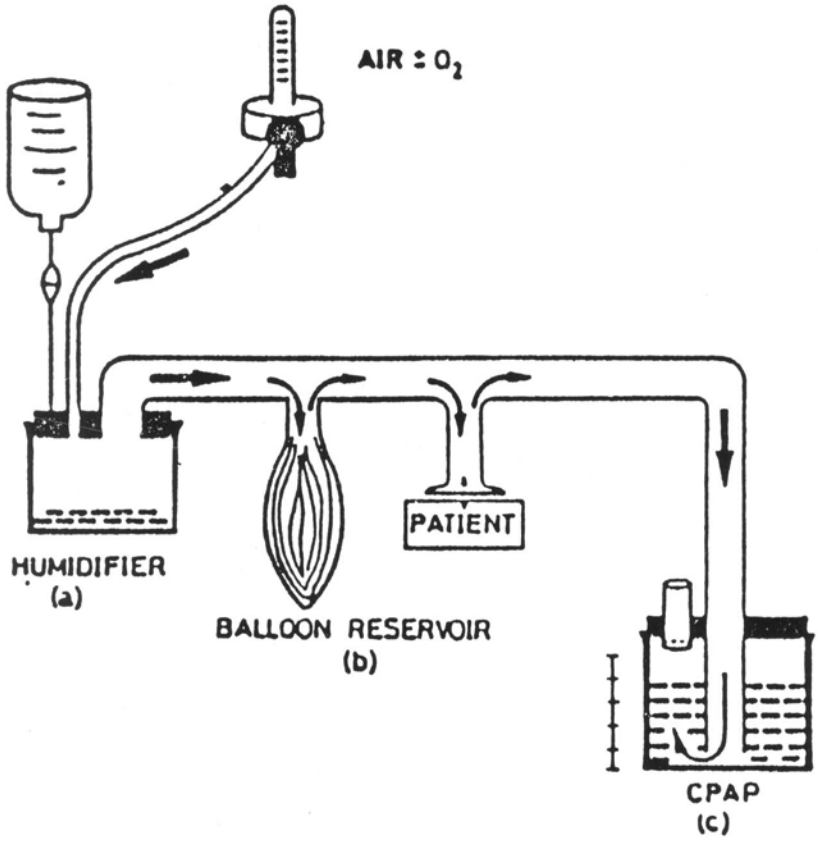


Fig. 1: The system consists of an air source with or without oxygen passing through a controlled heating and humidifying system (a) and connected to a balloon reservior (b). The amount of CPAP being controlled by the level of water in the exit chamber (c)

* Cardiac output.
CPAP Continuous positive airway pressure.
+5 & +10 cm indicate positive pressure.

c7 = c1-c2	p = 0.15	NS
c8 = c1-c3	p = 0.022	SG
c9 = c2-c3	p = 0.006	SG
c11= c4-c5	p = 0.11	NS
c10= c4-c6	p = 0.025	SG
c12= c5-c6	p = 0.095	SG

Discussion

It has been well documented that in mechanical ventilation there is increased risk of a fall in cardiac output which is further aggravated by positive end expiratory pressure (PEEP) if used. This has been studied extensively in man and experimental animals. The possible mechanism, which might be responsible for the alteration in cardiac performance during CMV + PEEP, was as follows. Cournand et al[4] assume that the primary cause would be reduction in venous return, resulting from increase in the intra-thoracic pressure. This will be more pronounced in hypovolumic patient. Another factor during CMV & PEEP was found to be an increase in pulmonary vascular resistance. Mead and Whittenberger[6] demonstrated this in dog lungs in Vivo. It has been assumed that an increase in pulmonary vascular resistance (PVR) would lead to right ventricular failure, thus decreasing cardiac output[7]. It has also been suggested that CMV + PEEP might, via the elevated intra-thoracic pressure, interfere with the coronary circulation leading to myocardial insufficiency[8]. Lately it has been suggested that the depressing influence on the myocardium might be mediated by a humoral agent, possibly released by stretching of the lung. In a study comparing CMV + PEEP, it has a significant increase in cardiac index with CPAP.

It was suggested that in a patient who is able to breath spontaneously, CPAP offers a very useful alternative to other methods of weaning as well as to CMV+PEEP. Our study showed that cardiac output did not decrease singnificantly after the application of CPAP + 5 and + 10 cm. Further more it was fairly well tolerated by the 'patients'.

CPAP is a well established and widely used technique in both adults and neonatal ventilatory support. The uncomplicated mechanical construction and the physiological nature of CPAP breathing make this treatment safe and easy to use. So far the possible indication of CPAP may be mild form of ARDS, prevention of pulmonary complication after major surgery. It can also be considered in patients with respiratory failure and cor pulmonale.

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