Hemodynamic Instability Due To Malfunction Of An 'Octopus' Connector

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Citation

G Girish. *Hemodynamic Instability Due To Malfunction Of An 'Octopus' Connector*. The Internet Journal of Anesthesiology. 2000 Volume 5 Number 2.

Abstract

Infusion of noradrenaline at low dose through a multilumen connector which had a malfunctioning non-return valve lead to hemodynamic instablity.

A 79 years old female patient with history of class 2 angina pectoris, shortness of breath and controlled hypertension presented with an acute abdomen. She underwent emergency sigmoid colectomy & colostomy for sigmoid colon perforation secondary to diverticulitis. Noradrenaline and dopexamine infusions were started intraoperatively to maintain the haemodynamics. Postoperatively the patient was transferred to the intensive care unit for overnight ventilation. She remained stable overnight and was extubated on the next day.

On the night of second postoperative day, the noradrenaline infusion (1.8 ml/hour of 4mg/40ml), in 50 ml syringe in a Graseby 3100 infusion pump, was connected to the largest port of the three-lumen octopus connector (Vygon, figure 1), which was in turn connected to one of the side ports of a triple lumen central venous catheter. Following this, the patient’s arterial blood pressure varied widely from 80 mm to 220 mm of Hg systolic and 45 mm to 90 mm Hg diastolic with an upshot approximately every10 minutes (figure 2 & 3). The central venous pressure varied from 5 to 9 mm Hg linearly with the arterial BP. Hypertension was accompanied by bradycardia and hypotension by tachycardia. Infusion pump failure was suspected but the fluctuation continued despite changing the infusion pump. The haemodynamic profile stabilised when the octopus connector was removed and the infusion was connected directly to the central venous catheter.

Figure 1

Figure 1 : The Octopus connector (multilumen extension tube) with non- return valves

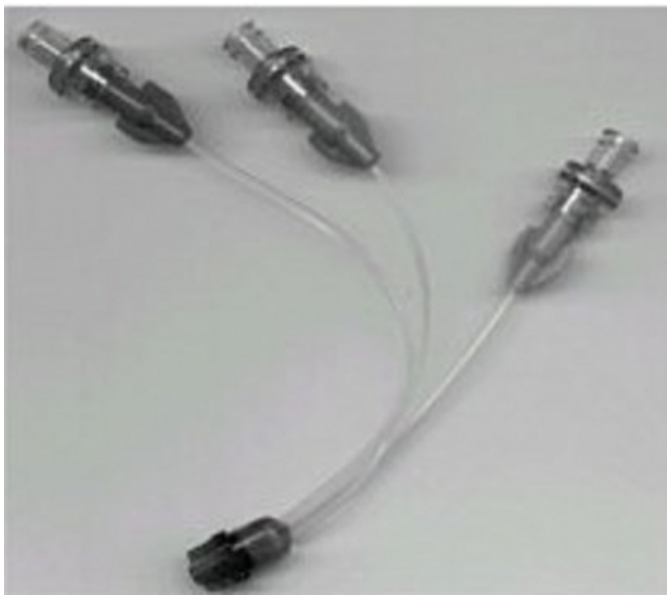


Figure 2

Figure 2 : The hemodynamic trend

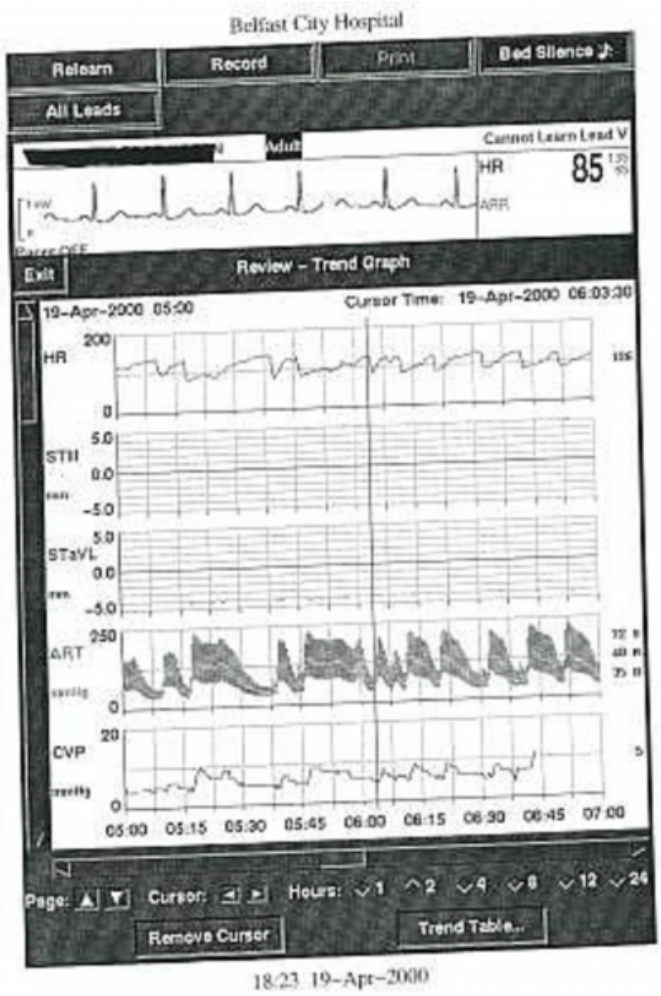
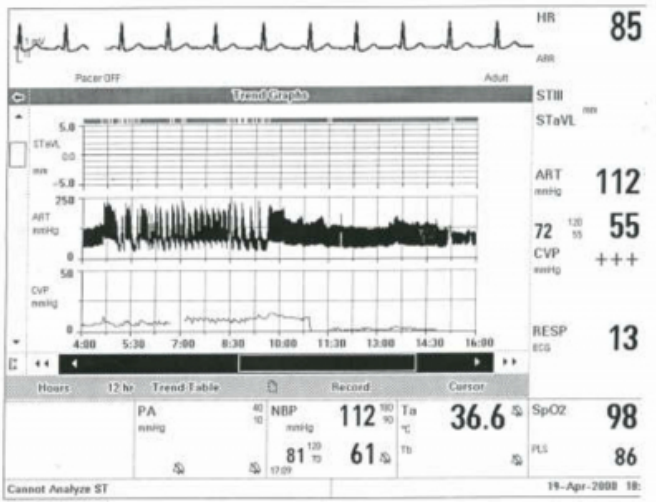


Figure 3

Figure 3. Trend Graph



There were no identifiable gross abnormalities in the octopus connector. A bedside test revealed intermittent obstruction to the flow through the connector, with a bolus (0.2 to 0.4 ml) delivery every 5-10 minutes.

The patient remained stable with no cardiac symptoms after this episode and was discharged from the intensive care unit on the following day.

DISCUSSION

Infusion flow rate has been shown to vary with the type of syringe pump, the volume of syringe, antisyphon device, flow rate, backpressure, solution viscosity, infusion rates and compliance of the extension tubing. Infusion pumps have been shown to be inaccurate at a flow rate of 1ml/hr ($_1$) and tend to be more accurate at 2ml/hr ($_2$). Changing the infusion pump did not make any difference to the blood pressure fluctuation in our patient. Stickiness of barrel of syringe has been shown to affect the flow continuity ($_3$). A standard 50ml syringe, which was filled to 40ml, was used for the infusion and the same syringe was continued throughout.

The time from pressing the start button until the initial flow for each Infusion set (start-up time) has been shown to be significantly longer with the antisyphon sets compared with the control when used at a rate of 2ml/hour. This difference minimized with higher flow rate ($_4$). The time interval in the hemodynamic fluctuation corresponded to that of flow as confirmed by the bedside test. The calculated volume of boluses (0.15-0.30 ml) was less than the dead space volume of the non-return valve (0.47 ml). The stickiness of the non-return valve might have caused intermittent obstruction to the infusion leading to marked hemodynamic variations

This equipment malfunction could have led to an adverse outcome for the patient. We will avoid using nonreturn valve and extension tubing sets with inotrope infusions at low rate

References

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