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TEMPERATURE CONTROL THERAPY SYSTEM FOR TREATING HEART ATTACK PATIENTS

I. OGHOGHO[1]

Department of Electrical and Information Engineering, Landmark University Omu-Aran, Kwara State, Nigeria
E-mail: oghogho.ikponmwosa@landmarkuniversity.edu.ng

ABSTRACT

Cardiac arrest with widespread cere-bral ischemia frequently leads to severe neurologic impairment. Recovery without residual neurologic damage after cardiac arrest with global cerebral ischemia is rare.Researchers have shown thattherapeutic hypothermia reduces brain damage and increases survivability in certain cases of cardiac arrest. Rapid infusion of cold intravenous fluid has been identified as the most effective means of inducing hypothermia. This work is targeted towards providing a device that is capable of inducing hypothermia on-spot and managing it automatically. An analogue to digital converter is used to monitor the output from a transducer depending on the patient's body temperature and give out digital outputs used to control when the thermoelectric module cools or heats the intravenous fluid and when it stays off. The system is designed to maintain the patient's temperature between 34°C and 36°C. A knob provided selects other temperature ranges. Different temperatures were generated to mimic body temperature being sensed by the transducer and the thermoelectric module's action was monitored. The intravenous fluid was heated when sensed temperature was greater than 34°C, cooled whenit was less than 36°C and neither heating nor cooling took place when it was between 34°C and 36°C. Although the system satisfied the fundamental operating principles, the rate of heat transfer to and from the intravenous fluid was poor. The intravenous tube also melted. These issues are being addressedin addition to making the system embedded. Biological life was not used for the test.

KEYWORDS: Temperature, cardiac arrest, emergency, therapy,control system

A heart attack occurs about every 20 seconds with a heart attack death about every minute (Allheartattack.com; 2011). An average of 1.5 million heart attacks occurs in the United States each year with 500,000 deaths(Allheartattack.com, 2011). One out of six deaths occurred in 2007 due to heart attack in the U.S. (AHA, 2011). An estimated 375,000 people in Europe undergo sudden cardiac arrest yearly (Jacqueline et al., 1997). Although the exact number of cases of cardiac arrest in Nigeria is not known, cases of patients who have suffered a heart attack is now on the increase thus giving health authorities deep concern. Cardiac arrest with widespread cere-bral ischemia frequently leads to severe neurologic impairment (The Hypothermia After Cardiac Arrest Study Group,2002). Recovery without residual neurologic damage after cardiac arrest with global cerebral ischemia is rare (The Hypothermia After Cardiac Arrest Study Group, 2002). Ischemia is a restriction in blood supply, generally due to factors in the blood vessels, with resultant damage or dysfunction of tissue. After cardiac arrest with no blood flow for more than five minutes, the generation of free radicals, together with other mediators, during reperfusion creates chemical cascades that result in cerebral injury (Negovsky et al., 1988)

Until recently, there was no therapy with documented efficacy in preventing brain damage after cardiac arrest. In the late 1950s, there were several case reports of the use of therapeutic hypothermia (TH) for neurologic injury after cardiac arrest, but it was subsequently abandoned because of the uncertain benefit and difficulties with its use (Resuscitation Central, 2010). Interest was rekindled in the early 1980s by Safar and colleagues at the University of Pittsburgh using the dog model (Resuscitation Central, 2010). Induced hypothermia (also known as therapeutic hypothermia) is the intentional induction of hypothermia for medical purposes. Such practice has been shown to reduce brain damage and increase survivability in certain cases of cardiac arrest (The Hypothermia After Cardiac Arrest Study Group, 2002;Resuscitation Central, 2010;Bernard et al.,2002; Erick et al., 2010)

Virginia Commonwealth University (VCU) Medical Center and the Richmond Ambulance Authority have developed a program that dramatically improves resuscitation and survival rates in cardiac arrest patients by starting therapeutic cooling early and ensuring it continues at the hospital (Cynthia, 2010) . They switched to the intravascular cooling method to try to get to the target temperature faster because they believed a lot of science

[1]**Corresponding author**

supports the theory that the faster you can get the temperature down to the time of the cardiac arrest, the better off that person is likely to be (Cynthia, 2010). The ARCTIC program centers on two main goals: to restart the heart as quickly as possible after cardiac arrest, and to protect the brain by starting cooling as very early; then bringing patients to a single specialized post-resuscitation facility. American College of Emergency Physicians also reported improved resuscitation and survival rates in cardiac arrest patients by starting therapeutic cooling early and ensuring it continues at the hospital hence; diverse array of physicians, nurses, and technicians in several different specialties all need to be on board in using therapeutic hypothermia as a cardiac arrest patient is transitioned from ambulance to emergency department to coronary care unit (Mitchel, 2009).

Recently, in patients randomly assigned to a hypothermia group for research, external cooling device was used to induce hypothermia for patients and if the target temperature was not reached on time then ice packs were required (The Hypothermia After Cardiac Arrest Study Group, 2002). Passive re-warming to a temperature above 36°C lasted for a median of 8 hours. In a previous study by a group of researchers (Bernard et al., 2002), cooling was induced more rapidly using intravenous fluid cooled with ice packs and for a shorter period than 8 hours.

This therapy is increasingly being used in hospital emergency departments and intensive care units and has called for advancement in technology to treat cardiac arrest patients. Induced hypothermia is usually accomplished by a combination of several methods: Rapid infusion of ice-cold intravenous fluids, cooling of internal organs, such as with nasogastric lavage with ice-cold water, evaporative cooling of the body surface, Intravascular cooling, using specialized vascular catheters, external cooling with ice packs or special cooling blankets and prevention of excess heat generation (fevers, shivering) using medication.Researchers have shown that an easy and convenient way to achieve a human core-temperature control is by infusion of intravenous fluid (Bernard et al., 2002). The covering of the patient with ice packs or cooling blankets during resuscitation is inconvenient for medical and nursing staff and the use of ice packs or refrigerated units (for forced air cooling) limit the use of these techniques to the hospital environment.

The use of intravenous fluid has more advantage than the other methods because the fluid enters the bloodstream directly thus having a direct effect on the body temperature. Fig1 shows the use of intravenous fluid to induce hypothermia. Most medical practitioners achieved this by putting the intravenous fluid in refrigerators and coolers just before infusion while monitoring the patient's body temperature. The oesophagus is the best advisable position to place the temperature sensing device.

This work is targeted towards modifying this research technique by providing a device that is capable of heating and cooling the intravenous fluid thus inducing hypothermia on-spot and managing it automatically as shown in fig.1. This research is limited to the design and construction of a temperature control device that is capable of inducing and managing hypothermia (34°C to 36°C). Two knobs are however provided that can be used to vary the setting as is required for temperatures outside this range.

MATERIALS AND METHODS

In order to achieve the aim set out, research on hypothermia therapy for treatment of heart attack patients were studied (The Hypothermia After Cardiac Arrest Study Group, 2002; Resuscitation Central, 2010; Mitchel, 2009; Bernard et al., 2002). Human body temperature control, general temperature monitoring and control systems as well as human body temperature control systems were also studied and various design options and their cost implications were considered (Wara,2009; Bassin et al., 2008). The system was broken down into four sections: The power supply unit, the sensing and control unit, the heating and cooling unit and the output display unit. The system was tested by generating different temperatures to mimic the human body temperature which was sensed by the transducer and the action of the thermoelectric module (heating the fluid to about 40°C or cooling the intravenous fluid to about 15°C or staying off when the monitored temperature is in the

required range) was monitored. The temperature transducer is to sense the temperature of the patient but no patient was used for the test because it would require the help of medical professionals and volunteers as patients or lab rats. The equipment however, was tested based on the underlying logical and electrical principles it was meant to perform.

Two thermometers were used; one monitors the temperature sensed by the transducer while the other monitors the temperature of the intravenous fluid after passing through the thermoelectric module. If the intravenous fluid was administered to a patient, it would have circulated through the patient's body and raised or lower the patient's body temperature depending on whether the module was switched to heat or cool the intravenous fluid. Since the body temperature of the patient is what is being monitored by the temperature sensor, the cooling or heating is done such that the body temperature of the patient is maintained between the chosen temperature range.

Principle of Operation of the System

The temperature sensor senses the core body temperature of the patient and sends output signals to the comparators whose other inputs have been set by the knobs provided at the upper and lower limit temperatures. There are three conditions to be considered:

(i) When the patient's body temperature is less than the lower limit temperature and the system is expected to heat the intravenous fluid. Comparator one (which monitors the lower limit temperature) gives a low output because it's +ve input voltage is less than it's -ve input voltage. This low output of the comparator is inverted by the NOT gate and used to switch relay L_1 from its normal ground connection to V_{CC}. Comparator two gives a high output because it's +ve input voltage is greater than it's -ve input voltage. This high output of comparator two is inverted by the NOT gate and hence the relay (L_3) is left in its normal position (contact is connected to ground).The output of the AND gate is low because one of its inputs is low hence the relay (L_2) remains in its normal condition (Connected to V_{cc}). Thus current flows through the thermoelectric module in the anti-clockwise direction as indicated in fig.2. This directional flow of current causes the heating of the intravenous fluid as required. LED1 switches on to indicate that heating is taking place while LED 2 and LED3 stay off.

(ii) When the patient's body temperature is in the desired temperature range and the system is expected to neither cool nor heat the intravenous fluid. In this condition both comparators give high outputs hence the output of the AND gate is high thus the contact of relay (L_2) is switched from Vcc to ground. Thus power is cut off from the thermoelectric module and neither heating nor cooling of the intravenous fluid takes place. LED2 switches on to indicate that neither cooling nor heating is taking place while LED1 and LED3 stay off.

(iii) When the patient's body temperature is above the upper temperature limit and the system is expected to cool the intravenous fluid. Comparator two (which monitors the upper limit temperature) gives a low output because its Non-inverting input voltage is less than its inverting input voltage. This low output of the comparator is inverted by the NOT gate and used to switch relay (L_3) from its normal ground connection to V_{CC}. Comparator one gives a high output because it's +ve input voltage is greater than it's -ve input voltage. This high output of comparator one is inverted by the NOT gate and hence the relay (L_1) is left in its normal position (contact is connected to ground). The output of the AND gate is low because one of its inputs is low hence the relay (L_2) remains in its normal condition (Connected to V_{cc}). Thus current flows through the thermoelectric module in the clockwise direction as indicated in Fig2. This directional flow of current causes the cooling of the intravenous fluid as required. LED3 switches on to indicate that cooling is taking place while LED 1 and LED 2 stay off.

RESULTS AND DISCUSSION

When the thermoelectric module heats the intravenous fluid, the temperature of the fluid after passing through the thermoelectric module was an average of 40°C for a fluid at normal room temperature. When the thermoelectric module cools the intravenous fluid, the temperature of the fluid after passing through the

module was an average of 15°C.

When the temperature sensed by the transducer is less than 34°C the thermoelectric module heats the intravenous fluid to 40°C before it is to be administered to the patient and the Red LED turns on. When the temperature sensed by the transducer is greater than 36°C the thermoelectric module cools the intravenous fluid to 15°C before it is to be administered to the patient and the blue LED turns on. When the temperature sensed by the transducer is between 34°C and 36°C the Green LED turns on, the thermoelectric module is turned off and neither heating nor cooling of the intravenous fluid takes place. Table 1 gives a summary of the results obtained.

The design was targeted to modify the research technique of putting the intravenous fluid in refrigerators and coolers just before infusion by providing a device that is capable of inducing hypothermia (that is forcing the patient's temperature to stay between 34°C- 36°C on-spot and managing it automatically. This helps to reduce damage done to brain cells after a heart attack has occurred.

The system satisfied the underlying electrical and electronic principles it was designed to perform (by cooling or heating the intravenous fluid or staying off within the required temperature range) but the heat transfer efficiency was found to be poor. It took a long period for a reasonable transfer of heat to or from the fluid to be achieved. The intravenous tube was also found to melt due to heat supplied to it.

Due to the issues noticed, the system is presently being modified to improve the efficiency of heat transfer. A non-poisonous metallic conductor with a large surface area will be used at the heat transfer interphase so that heat transfer to the intravenous fluid will be faster and more efficient. Eight thermoelectric modules making four compartments for transferring heat to and from the fluid are also presently being considered. The system will be made micro-controller based after which with the aid of medical personnel the system will be tested with Lab rats and subsequently human patients.

After addressing these issues, it is believe that the system will be practically ready to find application in therapeutic treatment for post cardiac-arrest patients. It can also be used in treatment of moderate hypothermia (which is often used for cerebral protection during anesthesia for cerebral aneurysm clipping) and treatment of Patients with hyperthermia.

Table1: Summary of Test Results

Temperature Sensed by the Transducer (^{o}c)	**LED Type turned on**	**Relay Action**	**Average Temperature of the intravenous fluid after passing through the module (^{o}C)**
<34	Red	The relay switches and heating begins	40
34 – 36	Green	Cuts off power to thermoelectric module	Same as the temperature before passing through the module
>36	Blue	The relay switches and cooling begins	15

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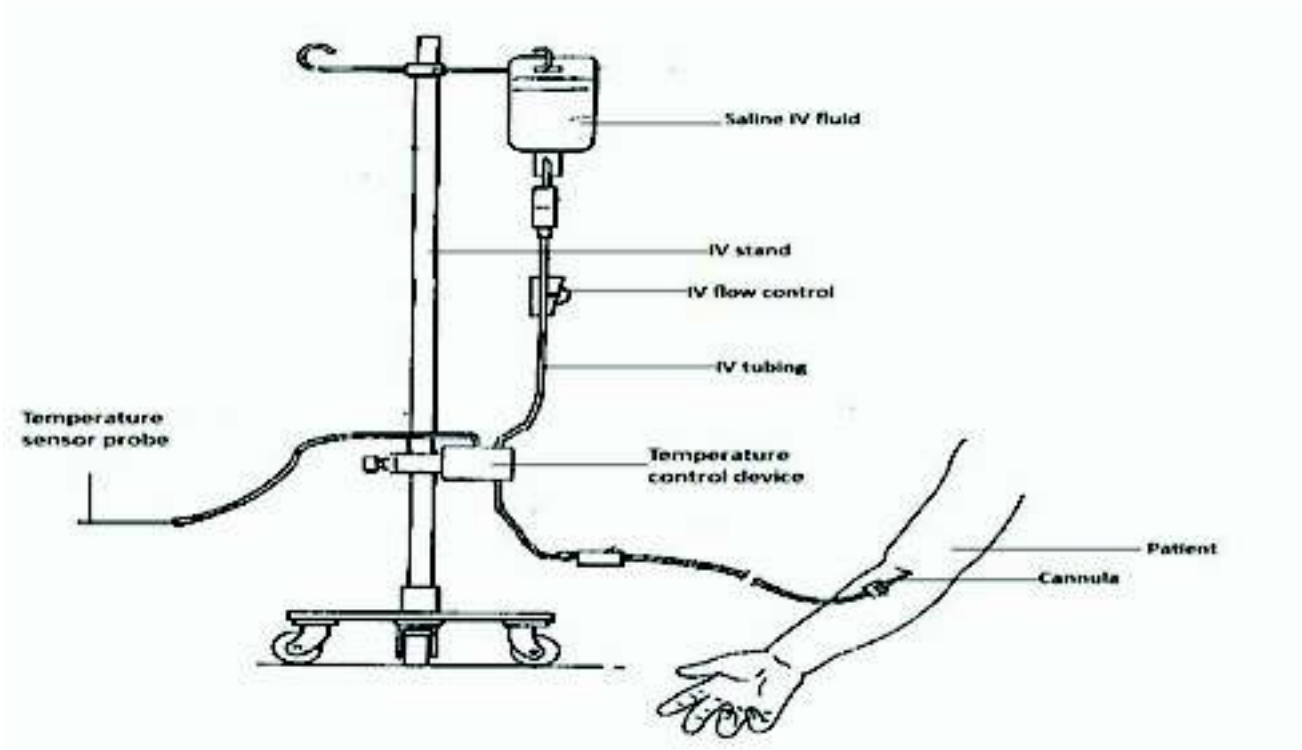

Figure 1 :Hypothermia being induced by rapid intrusion of intravenous fluid

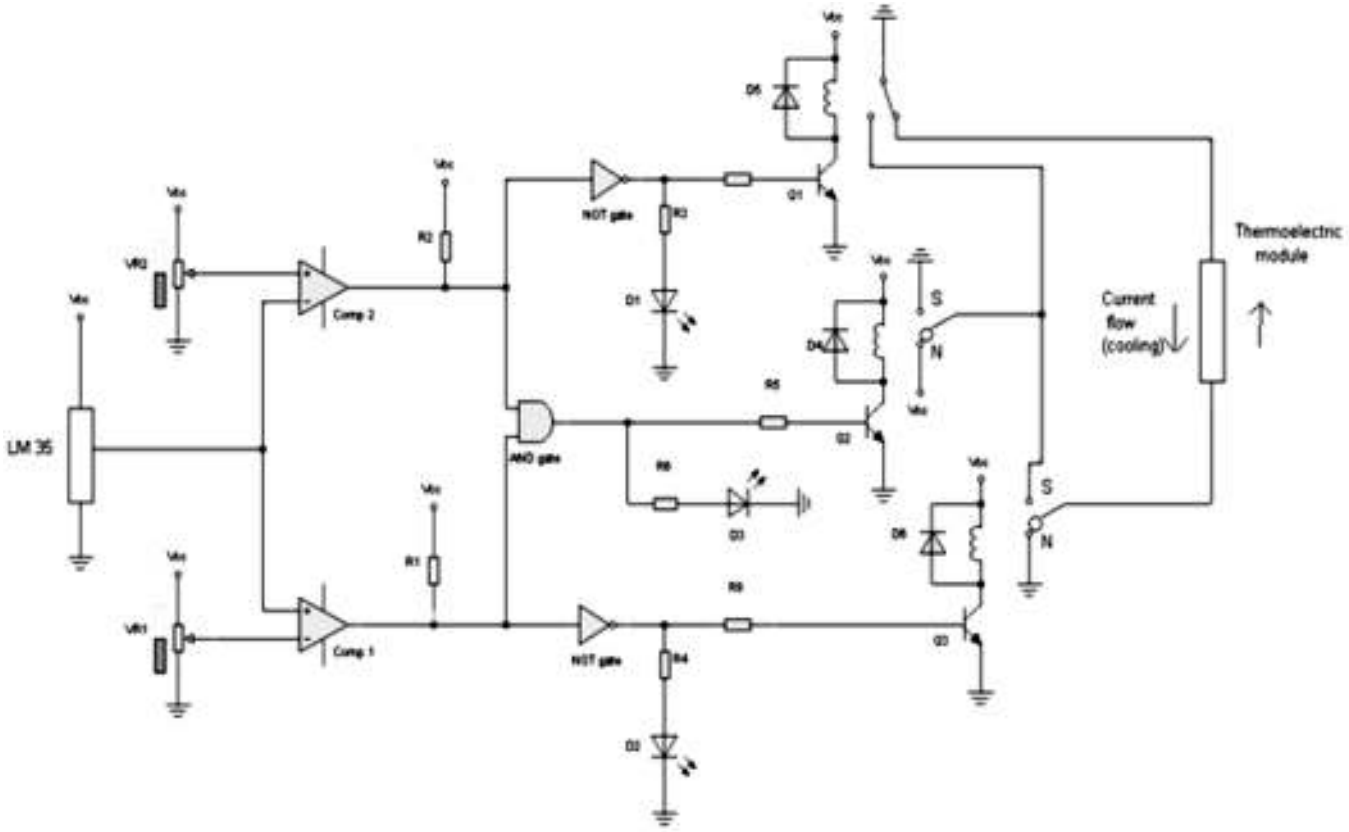

Figure 2: Circuit diagram of the Temperature Monitoring and Control System

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