

## Modification of a Low-pressure Alarm

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The need for low-pressure alarms to indicate ventilator failure as a result of mechanical fault or accidental disconnection from the patient has been described,<sup>1</sup> in a report of an alarm that has been used constantly at the Toronto General Hospital in the Respiratory Intensive Care Unit for several years and has proven to be very reliable. With the use of positive end-expiratory pressure (PEEP), it was necessary to redesign such alarms, since the constant pressure in the expiratory circuit of the ventilator prevented the alarm from being activated.

The alarm described has several safety features. There is no on/off switch so that the alarm may not be inadvertently left in the off position. A T-piece adaptor (fig. 1) placed close to the mouth ensures that the airway pressure monitored is likely to be that of the patient's airway and not the ventilator. A dependent water trap prevents the sensitivity of the alarm from being altered by condensation in the line from the humidified ventilator circuit. The suction-delay timer warns the nurse when suctioning is prolonged. Solid-state components chosen for reliability and low power consumption permit battery operation if desired. The whole unit can be economically assembled at a components cost of approximately \$100.00.

## DESCRIPTION

The unit (fig. 1) is approximately 5 inches wide  $\times$  7 inches high  $\times$  5½ inches deep. A wall-mounting bracket is provided on the back to permit location of the alarm near the ventilator and the patient. The sensor of the alarm is the electronic pressure transducer (fig. 2) that was developed in the Department of Anaesthesia at the Toronto General Hospital and was manufactured by the Medical Engineering Department of the hospital. The transducer is made by utilizing a strain gauge secured to a metal diaphragm and housed in a durable nylon enclosure.

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Received from the Department of Anaesthesia, University of Toronto, and Toronto General Hospital, Toronto, Ontario. Accepted for publication Oct 2, 1976.

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FIG. 1. The unit, with T-piece adaptor.

The pressure transducer, along with the circuitry, responds only to a pressure change, and hence, is not affected by PEEP. Consequently, a constant pressure (including zero pressure) caused by either disconnection or ventilator failure would cause the unit to alarm. The alarm circuitry resets at a change of 8 cm H<sub>2</sub>O, but the sensitivity is electronically adjustable and may be preset to sense any pressure change desired.



FIG. 2. Electronic pressure transducer.

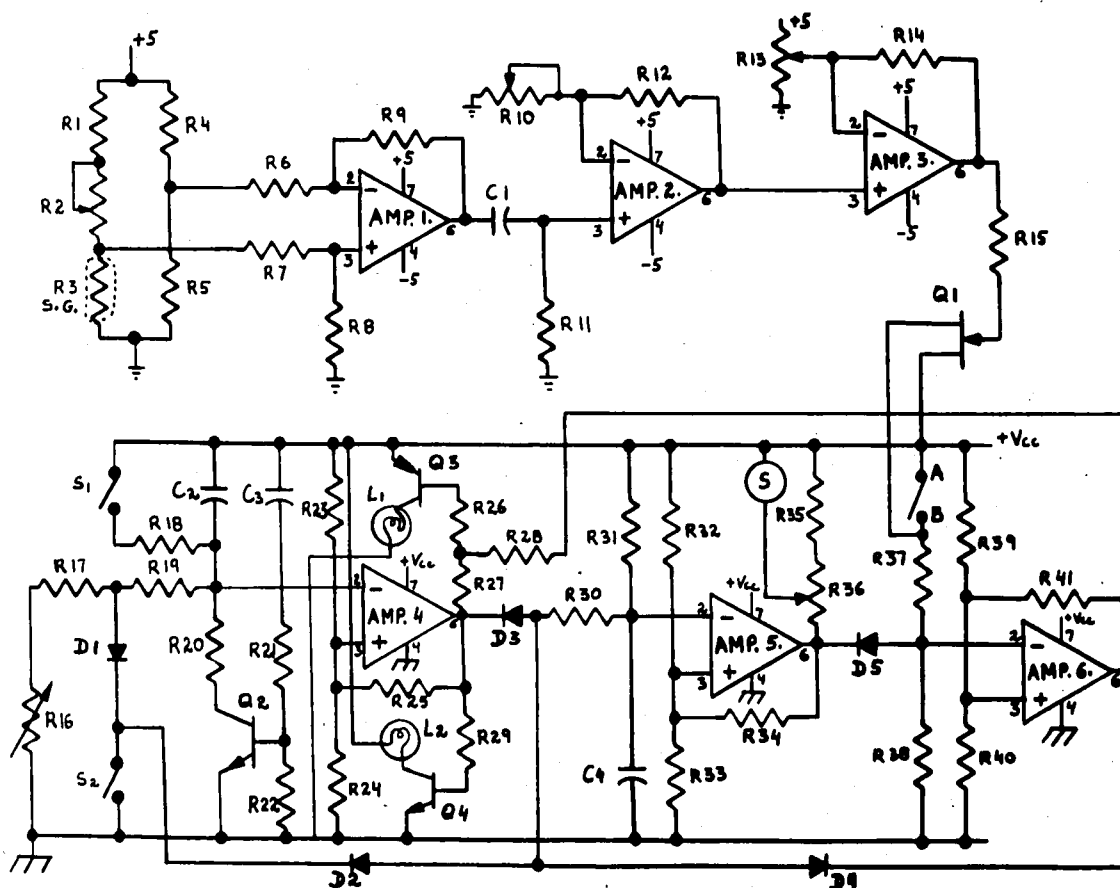


FIG. 3. Diagram of the alarm system.

Two indicator lights are provided on the front of the alarm. The light above the "reset" push button stays on so long as the alarm is plugged into the electrical supply. The "suction delay" push button suspends the alarm function (e.g., during tracheobronchial suction) for 30 seconds, and the suction-delay indicator light is illuminated for the period. When this time has elapsed, the alarm switches back automatically to its original state, and the alarm will sound if the pressure transducer does not sense a pressure change of 8 cm H<sub>2</sub>O within 10 seconds. In order to show that the alarm is functioning properly, the suction-delay light flashes every time the pressure of 8 cm H<sub>2</sub>O is exceeded.

#### The Circuit

A schematic diagram of the low-pressure alarm is shown in figure 3. Since the pressures measured are in the order of 8 cm H<sub>2</sub>O, in order to achieve maximum sensitivity, the strain gauge\* R<sub>3</sub> is placed in a Wheatstone Bridge configuration with resistors R<sub>1</sub>, R<sub>2</sub>, R<sub>4</sub>, and R<sub>5</sub>. The bridge is D-C excited

and should be balanced with R<sub>2</sub> while monitoring the output of the differential amplifier I with a differential gain of 35, using an oscilloscope. The following stage, AMP.2, is A-C-coupled with C<sub>1</sub> and R<sub>11</sub> (time constant approximately 5 seconds, to eliminate the D-C voltage caused by a constant pressure, e.g., PEEP), thus passing only the varying pressure signals. This varying pressure is amplified. The gain is adjusted by R<sub>10</sub> to approximately 1-volt peak with a change of 8 cm H<sub>2</sub>O. The output is coupled to a threshold comparator whose trigger point is preset by R<sub>13</sub>, thus controlling the junction FET, Q<sub>1</sub> as a switch.

Assuming that there is no pressure applied and Q<sub>1</sub> is off, amplifiers 4, 5, and 6 are saturated positively; therefore AMP.4 keeps the "reset" light L<sub>2</sub> on and the suction-delay light L<sub>1</sub> off and AMP.5 prevents the Sonalert(S) from sounding.

At the same time C<sub>4</sub> is charging through R<sub>31</sub>, and as inverting input voltage of AMP.5 exceeds the non-inverting input voltage set by the ratio of R<sub>32</sub> and R<sub>33</sub>, the AMP.5 switches negative and activates the Sonalert. The diode D<sub>5</sub> forces AMP.6 to remain in positive saturation, thus preventing AMP.6 from discharging C<sub>4</sub>, the timing capacitor,

\* Kulite Semiconductor Type DGP-1000-500.

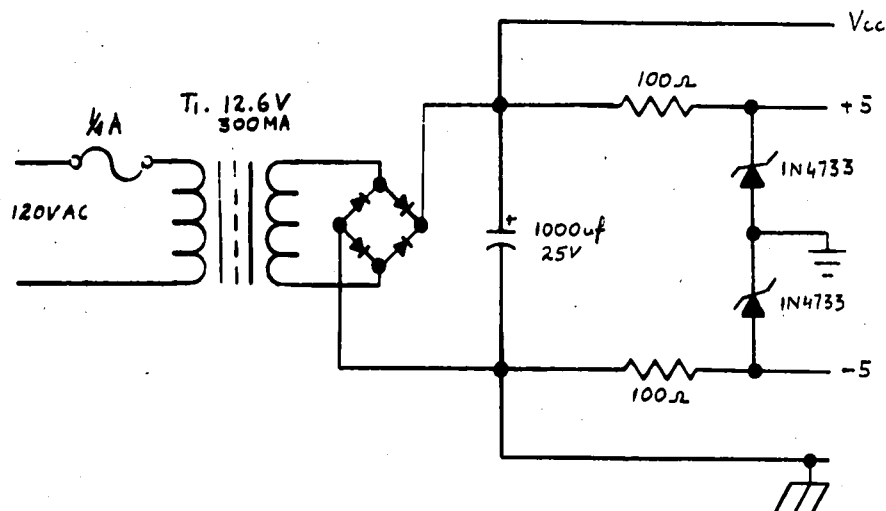


FIG. 4. Power supply circuit.

through  $D_4$  and  $R_{30}$ ; this would shut off the alarm before the emergency had been corrected. The only way the alarm may be reset is by manually depressing  $S_2$  and discharging  $C_4$  through  $D_2$  and  $R_{30}$ , deactivating the Sonalert. The potentiometer  $R_{36}$  is the loudness control of the Sonalert and  $R_{35}$  is the minimum limit.

Assuming the conducting state of  $Q_1$  when the pressure exceeds 8 cm  $H_2O$ , thus AMP.6 saturates negative and causing  $L_1$  to light up for the duration of inspiration. At that instant  $D_4$  is forward-biased, discharging  $C_4$  through  $R_{30}$  and preventing AMP.5 from changing state and sounding the Sonalert.

When suctioning of the patient is necessary, the alarm may be set in suction-delay mode by pressing  $S_1$ , which discharges  $C_2$  through  $R_{18}$ , thus causing AMP.4 to saturate negatively, disabling  $L_2$  and activating  $L_1$  (suction delay light). The output of AMP.4 also disables  $C_4$  from charging, since  $D_3$  is forward-biased. This shorts out the capacitor; therefore, the alarm cannot sound during suctioning. When  $S_1$  is released  $C_2$  begins to charge through  $R_{17}$  and  $R_{19}$ , forming a time delay of 30 seconds with  $R_{16}$  at minimum value. When this time has elapsed, AMP.4 will switch back to positive saturation, disabling  $L_1$  and enabling  $L_2$ . At the same time  $D_3$  is reverse-biased, resulting in  $C_4$  starting to charge, and the alarm will sound within 10 seconds unless enough pressure is applied within that time. The potentiometer  $R_{16}$  is an optional front panel control for use where suction delays longer than 30 seconds are needed.

When power is first applied, a current surge through  $Q_2$ ,  $C_3$ ,  $R_{21}$ , and  $R_{22}$  ensures that the alarm will always start in the short delay mode rather than suction delay by causing AMP.4 to negative-saturate temporarily, thereby discharging  $C_4$  through  $D_3$  and  $R_{30}$ .

If the PEEP feature is not necessary, the AMP.1, and AMP.2 and AMP.3, together with the associated components, may be eliminated, and  $Q_1$  may be replaced with a mechanical switch† between points A and B.

The power supply circuit is shown in figure 4. The circuitry incorporating AMPS.4, 5, and 6 was originally designed with a mechanical pressure switch. The power supply was single-ended 17 V DC, since the circuitry operates in a switching mode. When the PEEP feature was added, since the circuit consisted of linear amplifiers, it was necessary to divide the existing supply to  $\pm 5$  V DC using zener diodes. Note: There are two floating grounds not connected to line (instrument case) ground.

The circuitry described above was built on a single etched printed circuit board, mounted in a metal box. The power supply uses a  $\frac{1}{4}$ -amp. fast-blow fuse and a three-prong-plug power cord with the ground wire fastened to the case for safety. The alarm was Ontario Hydro-inspected and is approved for use with patients.

The parts list is appended.

#### APPENDIX 1

##### Parts List

- $R_1$ : 680 ohms,  $\frac{1}{2}$ w
- $R_2$ : 500 ohms, trim pot
- $R_3$ : 933 ohm  $\pm 2$  per cent strain gauge (gauge factor  $155 \pm 5$  per cent @ 75 F)
- $R_4, R_5, R_{18}, R_{19}, R_{27}, R_{30}$ : 1,000 ohms,  $\frac{1}{2}$  w
- $R_6, R_7, R_{20}, R_{35}$ : 2.7 kilohms,  $\frac{1}{2}$  w
- $R_8, R_9, R_{24}, R_{25}, R_{32}, R_{34}, R_{38}, R_{39}, R_{40}$ : 100 kilohms,  $\frac{1}{2}$  w
- $R_{10}$ : 5 kilohms, trim pot

† Dwyer Instruments Incorporated, Switch No. 1823-5.

R<sub>11</sub>: 33 kilohms, ½ w  
 R<sub>12</sub>, R<sub>15</sub>, R<sub>26</sub>, R<sub>29</sub>: 10 kilohms, ½ w  
 R<sub>13</sub>, R<sub>36</sub>: 10 kilohms, trim pot  
 R<sub>14</sub>: 1.5 megaohms, ½ w  
 R<sub>16</sub>: 2.5 megaohms, ½ w  
 R<sub>17</sub>: 500 kilohms, ½ w  
 R<sub>21</sub>, R<sub>22</sub>: 47 kilohms, ½ w  
 R<sub>23</sub>, R<sub>33</sub>, R<sub>41</sub>: 470 kilohms, ½ w  
 R<sub>28</sub>, R<sub>37</sub>: 4.7 kilohms, ½ w  
 R<sub>31</sub>: 1 megaohm, ½ w  
 C<sub>1</sub>: 100 μf, 16 v  
 C<sub>2</sub>: 22 μf, 35 v  
 C<sub>3</sub>: 10 μf, 35 v

C<sub>4</sub>: 4.7 μf, 35 v  
 D<sub>1</sub>-D<sub>3</sub>: 1 N 4154  
 Q<sub>1</sub>: TIS69 (N channel J.F.E.T.)  
 Q<sub>2</sub>, Q<sub>4</sub>: 2 N 3903  
 Q<sub>3</sub>: 2 N 3905  
 L<sub>1</sub>, L<sub>2</sub>: G.E. 344  
 S<sub>1</sub>, S<sub>2</sub>: Push button N.O.  
 S: Sonalert  
 AMP.1-AMP.6: μA 741

## REFERENCE

1. Lamont H, Fairley HB: A pressure-sensitive ventilator alarm. *ANESTHESIOLOGY* 26:359, 1965

Anesthesiology  
46:220-221, 1977

## Muscle Relaxants, Myasthenia, and Mustards?

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A search of the medical literature failed to reveal any connection between muscle diseases, neoplasms and the nitrogen mustards. This case suggests that the combination of nondepolarizing neuromuscular blocking drugs, alkylating drugs, and myasthenia gravis may be hazardous. In this case, the nitrogen mustard used was triethylene thiophosphoramide (thio-tepa, Lederle).

## REPORT OF A CASE

A 58-year-old woman, weighing 68 kg, was admitted for laparotomy, at which a malignant cystadenoma of the ovary was found, with metastatic seeding of the peritoneal cavity, omentum, bowel and liver. She had a positive diagnosis of myasthenia gravis, Group IIa,<sup>1</sup> of 6 months' duration, treated with pyridostigmine, 120 mg orally, *q.i.d.* Mild adult-onset diabetes mellitus was present, controlled by diet. Thyroid function was normal, clinically and biochemically. For anesthesia, the early morning dose of pyridostigmine was omitted, and atropine, 0.4 mg, im, given an hour preoperatively. A nerve stimulator (Block-Aid Monitor, BW), using needle electrodes along the ulnar nerve, was placed with the patient awake. While the patient was breathing 100 per cent oxygen, pancuronium bromide was injected iv, an initial dose of

0.5 mg, then increments of 0.25 mg at intervals of 1-2 minutes until the twitch response disappeared. The total dose was 1 mg. Then a sleep dose of thiopental was administered, and control of respiration achieved using N<sub>2</sub>O, 70 per cent in oxygen. After topical administration of 4 per cent lidocaine, the trachea was intubated with a cuffed endotracheal tube. Anesthesia was maintained with 70 per cent N<sub>2</sub>O, 30 per cent oxygen, and controlled ventilation with P<sub>A<sub>CO2</sub></sub> 35 torr. Muscular paralysis was monitored by single-twitch nerve stimulation. Pulse, blood pressure, ECG, arterial blood gases, and temperature were also monitored. After 50 minutes, the twitch response returned and slowly increased, but no more pancuronium was given. One and a half hours after the initial administration of pancuronium, thio-tepa, 60 mg, was instilled into the peritoneal cavity and allowed to remain. Within a minute, the twitch response disappeared. The instrument was checked for proper function. After a further 45 minutes, at the end of the operation, atropine, 1.2 mg, and neostigmine 5, mg, were administered iv, with no effect. The endotracheal tube was left in place and the patient was taken to the recovery room, where mechanical ventilation was instituted. Four hours after the intraperitoneal instillation, edrophonium, 20 mg, was given, without effect. Eight hours after this, there was a slight twitch response, and reversal with neostigmine was again attempted, but this also was not effective; gross fade and posttetanic facilitation were demonstrable. The next morning, the patient could open her eyes, but grip strength was only fair and the respiratory variables were borderline. Neostigmine was given again, the patient's condition improved considerably, and the tracheal tube was removed. Neostigmine was begun, 1 mg, im, every four hours. Because of fatigue,

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