Chapter 5
Cardiopulmonary Support: Evaluation And Intervention

Introduction

Since the first edition of this manual was written, Alcor has accumulated a significant amount of data about the effectiveness of CPR in suspension patients. Unfortunately, the indications are that CPR is failing to meet the metabolic needs of most suspension patients.

Some of the reasons for this were discussed in the preceding chapter: atherosclerotic disease, primary or secondary pulmonary disease (metastatic cancer of the lungs, pulmonary edema, COPD, etc.), and septic shock.

A review of resuscitation literature and Alcor's own unique clinical experience has served to expand our understanding of the limits of CPR administered during cryonic suspension.

Even under fairly optimum clinical conditions, where an otherwise healthy individual has experienced sudden cardiac arrest, direct measurement of mean arterial pressure (MAP) during *closed chest* CPR has demonstrated MAPs significantly below those known to be compatible with current criteria for cerebral viability (McDonald, J. *Annal. Emerg. Med.* 11:292-295, 1982). Evaluation of cerebral blood flows achieved in *healthy* dogs given CPR following induced ventricular fibrillation has demonstrated cerebral cortical blood flows which are only 19% of those observed prior to cardiac arrest and the initiation of closed chest CPR. By contrast, in the same study, open chest CPR was demonstrated to provide cerebral blood flows which were *67% of control.* (Tatsura, A., et al. *Resuscitation* 12:147-154, 1984).

The typical cryonic suspension patient to whom cardiopulmonary support can be applied is, with few exceptions and almost by definition, one who has experienced a long agonal course in a medical setting. (That is, one experiencing a "chronic death" as a result of a systemic illness which has impacted a variety of organ systems.)

Such a patient is *not* simply suffering from cardiac and respiratory arrest, but rather has a variety of underlying conditions which have *caused* cardiopulmonary arrest. The true proximate cause of cardiac arrest is thus likely to be any one or more of the following: shock due to dehydration, asphyxia as a result of pulmonary edema, acutely low blood sugar due to starvation or liver failure, sepsis with associated shock, hemorrhage due to tumor or stress related gastric ulcer disease, etc.

In short, the Transport Technician is presented with a patient who is often wasted, suffering from failure of a variety of homeostatic mechanisms, and is anything but "otherwise healthy". CPR will be even less effective in such a patient.

Alcor's clinical experience has borne this out. Evaluations of blood pH, glucose, and serum enzyme levels in several patients transported using CPR have demonstrated marked acidosis in spite of buffer administration (pH 6.9 - 7.0 vs. a normal of 7.4), very low blood glucose, (19-25 mg/dL vs. a normal of 70 - 110 mg/dL), and elevated serum levels of tissue enzymes such as LDH, CPK, SGOT, and SGPT. Low pH and whole blood glucose are indicative of poor perfusion, and elevated serum LDH, CPK, SGOT, and SGPT levels are associated with altered cell membrane permeability or actual cell lysis, presumably as a result of ischemia/hypoxia secondary to inadequate blood flow and oxygenation during HLR transport. (Unless pre-existing disease is responsible for their elevation.)

The Utility of CPR

All of the foregoing raises the question: "Then of what use is CPR?"

While CPR may not be delivering adequate blood flow or oxygenation over the time course of transport in most patients, it is delivering *some flow*. As a consequence, CPR is useful to provide some metabolic support (which we hope will become more effective as hypothermia is induced). More importantly, CPR does speed induction of hypothermia by increasing the effectiveness of external cooling, distributing medications which will hopefully mitigate ischemic (and reperfusion) injury, and distributing anticoagulants to prevent blood clotting that would seriously interfere with distribution of cryoprotective agents during subsequent perfusion.

CPR is still being used in suspension patients because it can be objectively demonstrated by evaluation of markers for injury (such as serum levels of tissue-specific enzymes) and by clinical signs (such as the integrity of the capillary bed during subsequent cryoprotective perfusion and the absence of clotting) that patients supported in this way remain in better condition than those who are not.

Clinical Assessment of CPR During Transport

The effectiveness of CPR in a given suspension patient can be expected to vary widely, depending upon the underlying cause of cardiac arrest and the agonal course. A patient dying of septic shock secondary to liver failure from metastatic cancer with complicating pulmonary edema can be expected to benefit far less than a patient who is pronounced legally dead as a result of an arrhythmia in a coronary care unit (followed by a brief and unsuccessful attempt at resuscitation).

Thus, it is of importance that the Transport Technician be able to assess the patient during CPR and arrive at an evaluation of its effectiveness.

Methods of Evaluation

The classical methods of evaluating the efficacy of CPR are the presence or absence of the carotid and/or femoral pulse, pupillary status, and skin color. Recent studies suggest that carbon dioxide excretion may be the most sensitive and useful indicator currently available for evaluating the efficacy of CPR (Sanders, A.B., et al., *JAMA* 262:1347, 1989.), (Gudipati, C.V., et al.,*Circulation* 77:234-239, 1988.).

The problem with the use of end-tidal carbon dioxide monitoring (measurement of the CO_2 concentration in the patient's expired air) has been the complexity, high cost, and bulkiness of *capnography* equipment required to reliably measure end tidal CO_2 in the field.

Early in 1989, Fenem Airway Management System began marketing a simple, inexpensive device which uses a sensitive, rapidly responsive chemical indicator to evaluate the carbon dioxide concentration in a patient's expired air during resuscitation or intubation efforts. The FEF end-tidal CO_2 detector is a simple, disposable device which is interposed between the endotracheal tube and the bag-valve unit or the HLR respirator hose. A nontoxic chemical indicator strip displayed beneath a transparent dome on the device detects CO_2 concentration with each breath. The device can measure CO_2 concentrations from 0.03% to 5% using three separate indicator strips.

Figure 5-1. *The FEF Detector in place between a bag-valve respirator and the patient's endotracheal tube.*

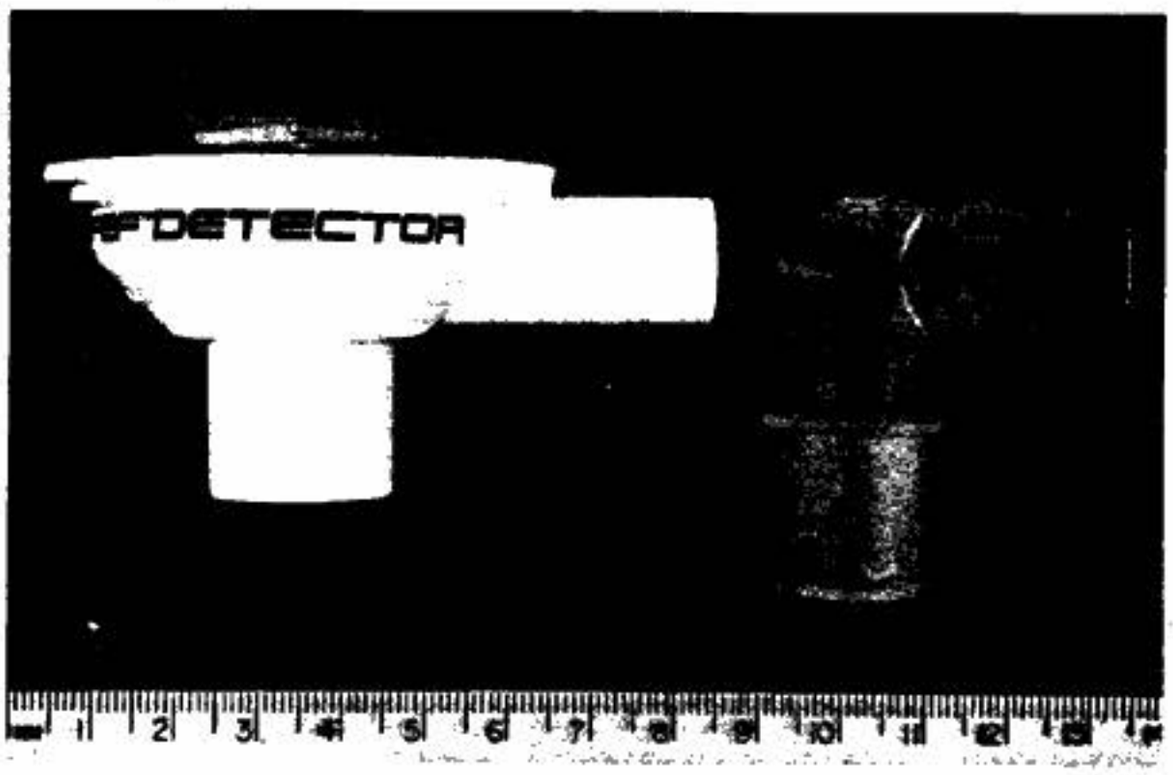
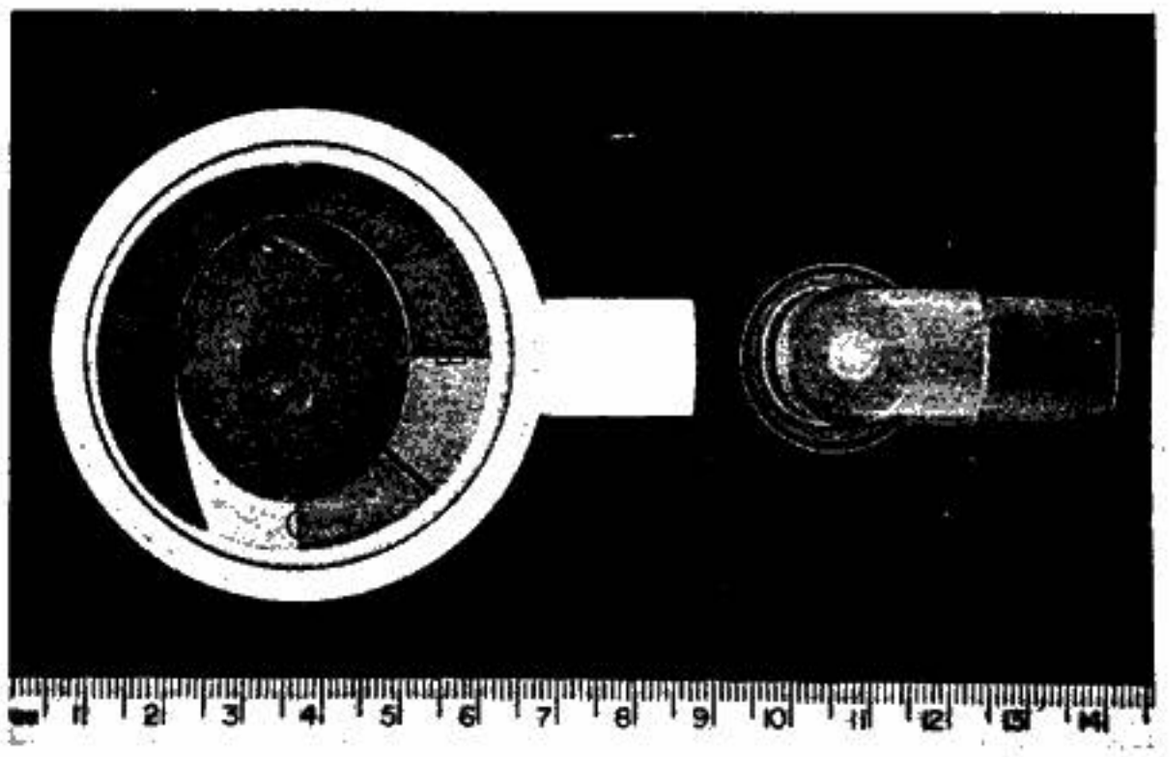


Figure 5-2. *The FEF end-tidal carbon dioxide detector in profile. A standard anesthetic circuit angle piece is shown for comparison.*

Figure 5-3. *Top view of the FEF detector, with a standard anesthetic circuit angle piece for comparison. Surrounding the clear window are areas marked* CHECK, A, B, *and* C *which represent a color chart of ranges of end-tidal CO_2 from room air (0.03% -- purple-*CHECK*) to 4% or greater (yellow-*C*).*



Conditions For Use of the FEF End-Tidal CO_2 Detector

The FEF Detector should be used from the very beginning of every transport procedure. Not only is the device a useful tool for evaluating the effectiveness (or lack thereof) of CPR, it is useful in determining if the endotracheal tube or EGTA has been placed correctly. Accidental intubation of the esophagus with the endotracheal tube, or

accidental placement of the EGTA obturator in the trachea will result in failed ventilation and thus in failure of gas exchange (i.e., oxygen will not be delivered to the lungs and carbon dioxide will not be carried away). The FEF Detector will thus show no difference in CO_2 concentration from inspiration to expiration.

The FEF Detector should be kept free of ice bags and should be protected from wetting. Cooling of the device can decrease its responsiveness. ASC has not yet had the opportunity to evaluate this device either in the field or in the laboratory in order to determine how it will behave in response to induction of hypothermia. Presumably, as the patient cools, CO_2 production will decline; and the responsiveness of the device may also be affected as the expired air stream coming from the patient decreases in temperature as well. Until these changes are documented and characterized in the laboratory, it will be difficult for the Transport Technician to assess the utility of the device over the time-course of an entire transport.

According to the manufacturer, the working life of the FEF Detector in a normal clinical situation is approximately 2 hours. In practice, many anesthesiologists using the device report that working life can be as long as 4 to 6 hours. In a typical HLR-assisted suspension patient transport the device may well last 6 to 8 hours.

In any event, it is anticipated that the period of peak utility for the device will be during the first 1 to 2 hours of cardiopulmonary support in order to optimize CPR, if possible, and document its effectiveness.

Procedure For Use Of The FEF Detector

1) Remove the FEF Detector from its aluminized mylar package. *Do not remove the end caps until ready to use the device.*

2) Match initial color of the indicator to the purple color labeled **"CHECK"** on the product dome. If the purple color isn't the same or darker, **DO NOT USE.**

3) Insert the endotracheal tube or EGTA. If an ET Tube is being used, inflate the cuff on ET tube. If an EGTA is being used, do not inflate the cuff on the EGTA obturator until correct placement of the obturator in the esophagus has been verified.

4) Remove end caps and firmly attach FEF Detector between the ET tube or EGTA mask and the breathing device.

5) Ventilate the patient with *six breaths* of moderate tidal volume. (This may be done slowly or quickly). **Note: Interpreting results with less than six breaths can yield false results since the stomach may initially contain some carbon dioxide.**

6) Compare the color of the indicator *at the end of the breath* to the color chart on the product dome.

7) The indicator color will continue to fluctuate from inspiration to expiration for up to two hours. Over time, due to varying patient humidity, lighting, or distance from the device, the color change on inspiration may not be as obvious or as detectable. The end-tidal CO_2 ranges, however, can still be detected at the end of the breath.

Optimizing CPR Using the FEF Detector

If evaluation of the patient using end-tidal CO_2 indicates inadequate perfusion (end-tidal CO_2 concentration is equal to or less than 0.5%) at the start of CPR, attempts should be made to identify the problem(s) and correct it. At least 3% CO_2 is preferred; 2% is the minimum acceptable. First, insure that the endotracheal tube (or EGTA obturator) is correctly positioned and that the patient is being adequately ventilated. If adequate chest expansion and gas delivery to the lungs is present, then attention should be given to the patient's hemodynamic status by insuring that the patient is adequately hydrated, and that vascular tone is sufficient to insure adequate perfusion. Keep in mind that transport medications typically will expand the patient's vascular volume by approximately 1 liter to 1,500 cc. Unless the patient is severely dehydrated, expansion of vascular volume by a liter should be sufficient.

If the patient's fluid status is determined to be adequate and end-tidal CO_2 concentrations are still not deemed to be adequate, attempt to secure adequate perfusion by administering pressor drugs to improve vascular tone. Phenylephrine HCl (Neo-Synephrine), 1%, 10 mg/ml should be administered intravenously. Following an initial bolus of 0.5 mg, observe the patient for a period of 10 minutes to determine if the drug has improved perfusion, as indicated by an increase in carbon dioxide concentration in the expired air. If there is no improvement in end-tidal CO_2, give a repeated bolus of medication.

If the patient does show a positive response to phenylephrine, establish an IV drip of the medication to provide on-going pressor support per the instructions below. Keep in mind that the half-life of phenylephrine under normothermic conditions is approximately 15 minutes. Hypothermia will decrease responsiveness to the medication and prolong its duration of effect by decreasing its rate of metabolism.

Procedure For Preparation And Administration Of Phenylephrine HCl

Bolus Administration

1) Withdraw 1cc (10 mg) of 1% solution from the ampule or multidose vial it is supplied in.

2) Dilute the medication by drawing up an additional 9 cc of normal saline, D5W, or other appropriate sterile diluent, for a total volume of 10 cc.

3) Administer 0.5 mg (0.5 cc) by IV push and observe the FEF detector for 10 minutes to determine if perfusion has been improved.

4) If no response is noted, administer another 0.5 mg (0.5 cc) dose by IV push.

Continuous Infusion

1) If the patient has responded favorably to the initial bolus of medication, a continuous IV drip should be established:

2) Inject 10 mg (1 cc) of phenylephrine 1% into a 500 cc bag or bottle of normal saline, D5W, or other appropriate IV solution. Thoroughly mix the added medication with the vehicle solution.

3) Begin a continuous IV infusion at a rate of 40 to 60 drops per minute. Adjust the dose (between 40 and 60 drops/min) on the basis of the patient's response.

Clinical Methods

All three of the clinical (i.e., those not requiring special measuring equipment) methods of patient assessment listed under *Methods of Evaluation* should be used by the Transport Technician. However, three caveats apply:

1) The presence of a carotid and/or femoral pulse is a *necessary but not sufficient* sign of adequate CPR. CPR may be effective at generating a palpable pulse, but the duration of the high pressure wave (the MAP) and the pressure on the venous side of the circulatory system (which is markedly elevated during closed chest CPR) determine *actual blood flow*. Thus, it may be possible to have a peak arterial pressure of 160 mmHg (normal is 120 mmHg) and still have almost no blood *flow* since the *time* spent at this pressure may be very short and the venous pressure may be markedly elevated.

2) Reactivity of the pupils to light is unlikely to return during closed chest CPR, even when administered to patients who have suffered cardiac arrest without associated complicating pathologies. In most cases, CPR is simply not effective enough in meeting cerebral metabolic demands to allow for recovery of pupillary reactivity. The administration of potassium chloride and high doses of barbiturates will also result in pupils which are fixed in mid-position.

3) Of the three clinical signs, skin color is probably the most reliable, since a return of color to skin and mucosa indicates delivery of oxygen-saturated blood, at least to those tissues. However, this sign may also be misleading since the skin will be the first to cool and will thus experience reduced metabolic demands (reflected by a failure of even the very poor flow of oxygenated blood being delivered by CPR to be de-saturated). However, it is still useful to note a return of color, even if it occurs late in CPR and external cooling, since it is indicative of the delivery of *some* oxygenated blood to the tissues.

Despite the limitations discussed above, careful documention (note-taking) of these clinical signs in the patient's transport record is important.

Other Clinical Signs

Because CPR during transport operations is carried out for a period of *hours* rather than the clinical norm of 15 to 30 minutes, the Transport Technician may encounter complications not documented in the medical literature which are of relevance in evaluating the efficacy of CPR. Two of these signs, representing serious complications, repeatedly ahve been experienced during suspension operations by ASC personnel: fulminating pulmonary edema, as indicated by a frothy pink pulmonary exudate seen in the endotracheal tube, and massive gastric hemorrhage, as indicated by the leakage of blood/stomach contents from the mouth or gastric tube of the EGTA during resuscitation.

Pulmonary edema may be a pre-existing condition in the patient. But regardless of whether some degree of pulmonary edema preceded transport, pulmonary edema occurs frequently and with rapid onset (<30 minutes) of the start of closed chest CPR. This is because closed chest CPR results in high intrathoracic pressures with intra-cardiac pressures generated on the down-stroke being equal *in all four chambers of the heart*. The combination of low cardiac output and high pulmonary venous pressure is a recipe for pulmonary edema. Research has documented the *rapid* development of pulmonary edema and resultant poor gas exchange in humans receiving close chest CPR (Ornato, et al., *Crit. Care Med.* 11:79-82, 1983). One study that examined 2228 unsuccessfully resuscitated cases of prehospital cardiac arrest documented a 46% incidence of pulmonary edema at autopsy

(Nagel, E.L., et al., *Crit. Care Med.* 9:424, 1981).

The Transport Technician should thus be alert to the development of clinical signs of pulmonary edema such as gurgling noises during ventilation, or the presence of a pulmonary exudate (usually blood-tinged, light pink in color) welling up in the endotracheal tube or in the mask of the EGTA. Frequent auscultation of the patient's chest during transport is recommended so that the development of pulmonary edema can be determined as soon as possible and documented in the patient's record. The development of rales before the presence of pulmonary exudate is noted in the endotracheal tube also offers the possibility of intervention with aggressive suctioning, thus protecting what little gas exchange capability may remain.

Gastric hemorrhage during cardiopulmonary support has been noted as early as an hour after the start of CPR on two occasions in patients without known pre-existing ulcer disease. Several possible causes for this bleeding have been put forward:

1) Injury to the gastric mucosa may occur during the agonal period when the patient is hypoxic and in shock. Patients experiencing chronic death may remain in a state of deep shock for hours, resulting in greatly reduced or absent blood flow to the digestive tract and limbs. Interruption of normal blood flow to the stomach will reduce the pH buffering and osmotic regulation of the gastric mucosa normally provided by the blood flowing though it. Hypoperfusion of the gastric mucosa, also, probably results in decreased secretion of protective mucous and prostaglandins, further increasing mucosal vulnerability to erosion by gastric contents.

Much of the insult to the gastric mucosa may thus result from events occurring *before* the declaration of legal death.

2) Inadequate blood flow during CPR (*after* legal death), combined with massive anticoagulation secondary to heparin administration, may initiate erosion of the gastric mucosa and precipitate hemorrhage or exacerbate injury which is already present.

If it occurs, it is important to note the presence of gastric hemorrhage as evidenced by dark blood with or without stomach contents leaking from the mouth or the gastric tube. Filling of the stomach with blood (as evidenced by oral leakage) is indicative of the loss of a significant volume of blood and the probable total compromise of CPR.

If gastric hemorrhage is observed, the time it occurred and a description of the hemorrhagic fluid should be entered on the patient's *Transport Data Collection Sheet*.

Rigor Mortis has been observed in the jaw muscles within 2-3 hours of the start of CPR during the care of two patients. Rigor is likely a result of inadequate perfusion of the muscle and low blood glucose levels.

The patient's jaws and limbs should be evaluated periodically during CPR support to determine the time of onset and rate of progression of rigor.

Laboratory Methods

A variety of laboratory methods are available to evaluate the effectiveness of CPR in meeting the patient's metabolic demands. Some of these, such as measurement of serum lactate and tissue-specific enzyme levels, cannot be applied in the field. Nevertheless, they are relevant to the transport technician in that subsequent evaluation of these

markers for injury will *not* be possible unless blood samples are collected at regular intervals during the transport procedure (See *Chapter 13: Collection of Blood Samples*). Therefore the Transport Technician must be diligent in collecting these samples.

As data from previous suspensions indicates, blood glucose becomes very low during sustained CPR. This probably occurs as a direct result of the ineffectiveness of CPR in meeting the patient's metabolic demands. Under normal circumstances, the liver and pancreas regulate the concentration of blood glucose. Low blood glucose during CPR is almost certainly a result of failure of the liver to produce glucose (due to inadequate hepatic perfusion) and of the consumption of available glucose stores by anaerobic metabolism (also as a result of inadequate perfusion).

Regardless of the cause, blood glucose levels in the 19 to 25 mg/dL range (60-70 mg/dL is normal) are cause for serious concern whether or not they are directly indicative of the inadequacy of CPR. Fortunately, blood glucose may be simply and reliably measured in the field using any of a variety of reagent strips and a whole blood sample collected from the patient.

The system Alcor has selected for the evaluation of blood glucose during field transport of patients is the *Chemstrip bG* and the *Tracer II* blood glucose monitor, both of which are manufactured by Boeringer Mannheim Diagnostics. The procedure for use of the Chemstrip bG is detailed below, and the operating instructions for use of the Tracer II appears at the ends of this chapter in the form of the manufacturer's instruction booklet. Refer to *Chapter 13: Collection and Evaluation of Blood Samples* for blood collection technique.

Procedure For Use Of The Chemstrip bG

Whole blood glucose should be evaluated as soon after the start of cardiopulmonary support as is possible and at 30 minute intervals *or as necessary to assure that the blood glucose levels are no lower than 80 mg/dL and no higher than 120 mg/dL*, as follows:

1) Assemble the necessary materials: a vial of Chemstrip bG test strips, a gauze square or cotton ball, and a stop watch or wrist watch with second hand or timer feature.

2) Remove the Chemstrip bG test strip from the vial (and immediately recap the vial).

3) Obtain a venous blood sample.

4) Deposit a large droplet of blood on the test pad of the strip *completely covering it. Do not smear the blood on the test pad.*

5) Immediately begin timing for 60 seconds.

6) When 60 seconds have lapsed, wipe the blood from the test pad of the strip using moderate pressure with a cotton ball or gauze square. Lightly wipe the strip twice more using the clean side of the cotton ball or gauze square. Continue timing for an additional 60 seconds. *Do not stop timer after the first 60 seconds. Continue to time while you wipe the strip.*

7) After waiting the additional 60 seconds (total time elapsed: 2 minutes), match the colors on the test strips to the color scale on the vial label. If the colors

developed are darker than those for 240 mg/dL, wait an additional 60 seconds (total elapsed time 3 minutes) before comparing the final reacted colors to the color scale.

NOTE: Intermediate values can be estimated when the reacted colors fall between those on the vial label. For example, if the lower reacted test pad matches the lower pad 80 on the vial, and the upper reacted test pad matches the upper pad 120 on the vial, the estimated value is 100.

Reacted Chemstrip bGs should be saved, secured to the patient's *Transport Data Collection Sheet*, and returned to Alcor with the patient.

Never use expired strips. It is the Transport Technician's responsibility to keep Chemstrips up to date. Replacements may be purchased at any pharmacy without a prescription.

Adjustment of the Patient's Blood Glucose

If the patient's blood glucose is at or below 60 mg/dL, intervention to raise it is necessary. Typically, each 50 cc of 50% dextrose (which is the same thing as glucose) will raise the blood sugar of the average 72 kg man by 100 mg/dL. Thus, each 1 cc of 50% dextrose will increase the patient's blood sugar by 2 mg/dL. The dose in cc's of 50% dextrose administered should thus be determined by subtracting the patient's measured blood glucose from the desired blood glucose and dividing by 2, thus:

Dose 50% dextrose (ml) = (req. glucose (mg/dL) - meas. glucose (mg/dL)) ÷ 2

Once the dosage of dextrose has been calculated and drawn up, it should be administered by IV infusion using the flash bulb or medication addition port on the IV set.

Fifteen minutes after the administration of glucose, the patient's blood glucose should be re-evaluated and additional glucose administered as indicated. *It is important not to overshoot and increase blood glucose significantly above 120 mg/dL, as elevated levels of blood glucose during CPR are associated with increased cerebral injury.*