



## **Pediatric Anesthesia & Pain Management**

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**Anesthetic Management of the Diabetic Child**

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# Anesthetic Management Of The Diabetic Child

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# Anesthetic Management Of The Diabetic Child

## Introduction

To manage a child with juvenile onset (Type I) diabetes mellitus undergoing surgery, there are 2 important goals and 3 important principles that the anesthesiologist should keep in focus. These are as follows:

### ***Goals of Anesthetic Management of the Diabetic***

1. Minimize physiologic stress, thereby minimizing the propensity to develop diabetic ketoacidosis, or DKA.
2. Maintain euglycemia, thereby minimizing the risk of postoperative infection and maximizing the rate of wound healing.

### ***Principles of Managing the Diabetic***

1. Hyperglycemia reflects the dehydration/hypovolemia, not the adequacy of insulin therapy.
2. When serum glucose is over 300 mg/dl, hyperglycemia is inversely proportional to renal function, that is, the higher the serum glucose, the lower the creatinine clearance.
3. Therefore, the adequacy of insulin therapy cannot be judged from the blood sugar. Rather, the insulin dose should be determined by the magnitude of stress, and the acid base status.

These goals and principles will be explained and discussed below. In order to understand the basis for managing diabetics, it is first useful to examine the endocrinology and physiology of the worst case scenario, DKA.

## **The Role of Insulin in the Body:**

Insulin is the body's primary anabolic hormone, that is, the role of insulin is the facilitation of the storage of energy molecules in the body during normal physiologic states, in order to have them available during periods of stress and/or starvation. Therefore, in the absence of insulin, there will be

1. Inhibition of lipogenesis, and accelerated lipolysis, with the resultant release of free fatty acids into the circulation. These fatty acids can then be converted in the liver to ketoacids, a substrate that can be used by the brain, heart, and red blood cells as an energy source.
2. Inhibition of protein synthesis, and accelerated proteolysis, with resultant release of amino acids into the circulation. Some of the amino acids are then converted in the liver to glucose (gluconeogenesis).
3. Inhibition of glycogen synthesis in liver and skeletal muscle, and accelerated glycogenolysis, with resultant release of glucose into the circulation.
4. Inhibition of glucose uptake at the cellular level except in brain, heart, and erythrocytes, with resultant inhibition of glucose metabolism and utilization.

## **The Causes of DKA**

More than just a surfeit of insulin is required to result in DKA. There must also be excessive activity of the glucose counter-regulatory hormones, known in our specialty as "stress hormones." These hormones are:

1. Glucagon.

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2. Epinephrine.
3. Cortisol.
4. Growth hormone.

In the normal circumstance the balance between insulin and stress hormones both during fasts and after meals, in order to maintain a well regulated level of glucose in the blood, and to allow either storage of excess glucose, or production of needed glucose and other substrates (e.g. ketoacids) during fasting. In the diabetic child, DKA results when both insulin is lacking AND when the stress hormones are overactive. Lack of insulin alone will only produce hyperglycemia, the stress hormones are an absolute condition for the production of DKA.

Some of the common causes of over activity of the stress hormones are:

1. Trauma and burns
2. Infection.
3. Surgery.
4. Dehydration.
5. Pain and stress.

The importance of the role of the anesthesiologist in the prevention of DKA in diabetic children therefore becomes evident.

### **The Physiologic and Biochemical Derangements in DKA.**

The principal derangements during DKA are:

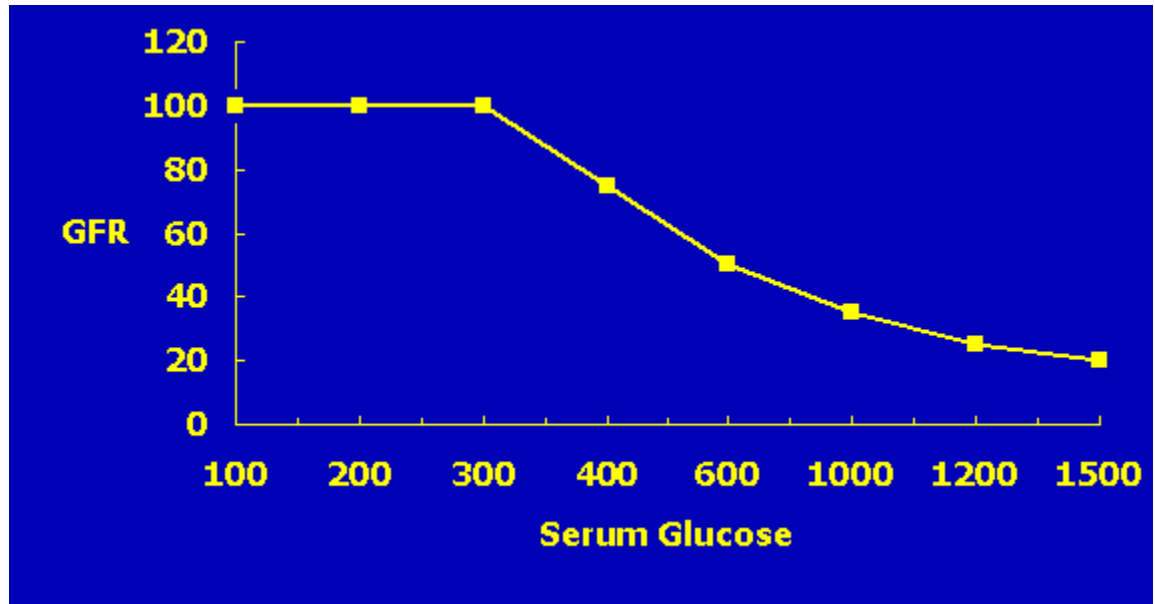
1. Hypertonicity.
2. Electrolyte losses.
3. Metabolic acidosis.
4. Hypocapnia.

Each of these derangements, in turn, results in many secondary effects:

#### ***Hypertonicity & Hyperglycemia:***

Hypertonicity is caused by (a) free water losses, and (b) hyperglycemia. The magnitude of the former is frequently overlooked, but bear in mind that during DKA, excessive quantities of urine are excreted as a result of the osmotic diuresis, and that the urine sodium content is approximately 50-90 mEq/L, e.g., it is about 1/2N saline. Therefore, water is being lost in excess of sodium. Volume contraction occurs, and this in turn leads to renal impairment, less glucose excretion, and further exacerbation of the hyperglycemia. In fact assuming there is no exogenous glucose source, when the blood sugar is in excess of 300 mg/dl, the most important determinant of the blood sugar is the creatinine clearance, not the amount of insulin being excreted or administered:

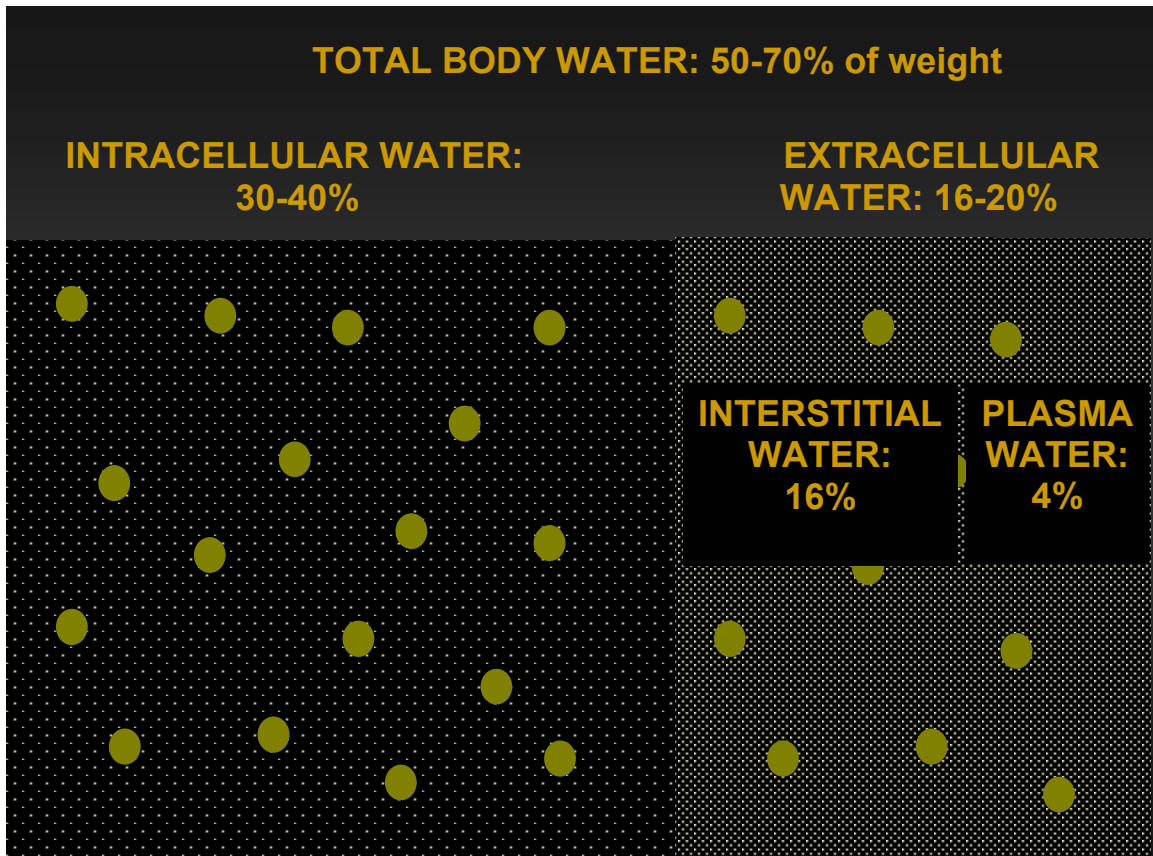
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**Figure 1.** *The relationship of serum glucose and GFR.*

Hyperglycemia also produces cellular dehydration in excess of extracellular dehydration because of the establishment of a hypertonic state. This means that the traditional cardiovascular signs of dehydration and hypovolemia will significantly underestimate the magnitude of free water losses during DKA.

The mechanism of this is that the hypertonicity draws free water out of the interior of the cells, and into the extracellular space (both the intravascular space, and interstitial or “third” space (Figures 2 and 3).



**Figure 2.** The normal distribution of water in the body. Large dots depict glucose molecules, evenly distributed between ICW and ECW. Small dots depict sodium ions.

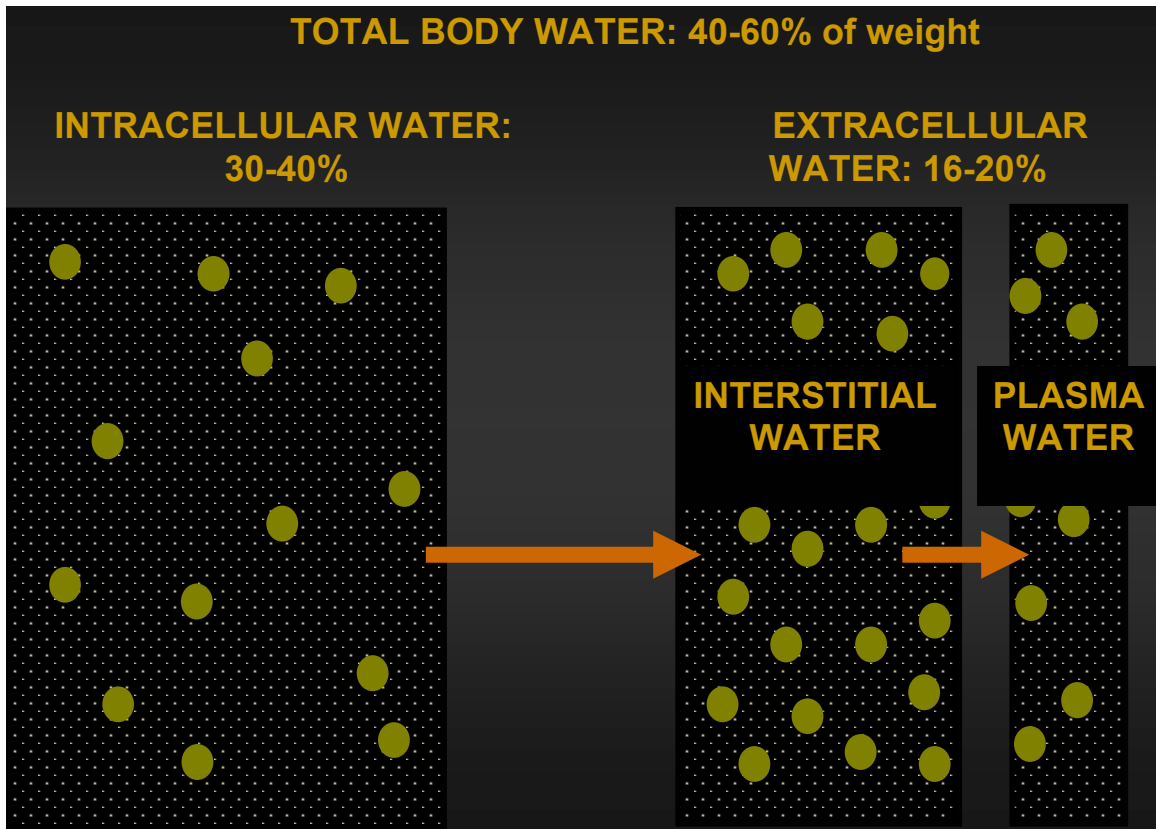
The important corollaries of these points are:

1. The magnitude of hyperglycemia is proportional to the magnitude of dehydration (provided there is no exogenous glucose source).
2. Therefore, serum glucose will fall merely by rehydrating the patient, and without administration of insulin.
3. Therefore, the serum glucose is not a good measure of the adequacy of insulin therapy.

### ***Electrolyte losses, metabolic acidosis, and hypocapnia***

Hyperglycemia produces an osmotic diuresis that results in passive electrolyte losses. Often diabetics in DKA have vomiting which aggravates the losses of electrolytes. The typical deficiencies in DKA are as follows:

1. Water, 10-15 ml/kg (e.g. 10-15% dehydration)
2. Sodium, 5-7 mEq/kg
3. Potassium, 4 mEq/kg
4. Phosphate, 4 mEq/kg



**Figure 3.** During DKA, there is a net loss of water from the body, most of which is lost from the ICW. The increase in glucose in the ECW produces hypertonicity dilution of sodium in the ECW.

**Sodium** is lost by osmotic diuresis and because of obligate cation losses accompanying excretion of ketoacid anions. In spite of the fact that 1/2N saline has been lost in the urine and that water losses exceed sodium losses, the serum sodium is not elevated as would be expected, but rather is lower than normal. This is due to the movement of water from the ICW to the ECW as illustrated in Figure 2, and dilution of the sodium in the ECW. The magnitude of this sodium depression can be easily estimated by knowledge of the serum glucose, such that for every elevation of serum glucose of 100 mg/dl, the sodium will be depressed by 1.6 mEq/L:

$$Na_{true} \approx Na_{measured} + 1.6 \cdot \frac{(Glucose - 100)}{100}$$

**Potassium** is lost also by osmotic diuresis and due to the action of aldosterone. In spite of large potassium losses in DKA, the serum potassium is usually elevated because of the shift of potassium from the ICW to ECW due to acidosis. This is important to remember because unless supplemental potassium is administered during DKA, the serum potassium will fall dangerously during correction of the acidosis.

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**Phosphate** losses can be substantial but have not been linked with any clinical problems. Administration of phosphate can precipitate hypocalcemia, therefore routine phosphate therapy is no longer recommended.

**Metabolic acidosis** results from the hepatic production of ketoacids (beta-hydroxybutyric and acetoacetic acids), as well as lactic acidosis. The acidosis will correct without specific therapy by restoring circulating blood volume and inhibiting hepatic ketoacid synthesis by administration of insulin. Therapy with bicarbonate is almost never warranted, and will result in precipitation of hypokalemia, arrhythmias, and paradoxical CNS acidosis. Secondary **hypocapnia** will slowly resolve with resolution of acidosis.

### Treatment of DKA in surgical patients

In the nonketotic diabetic patient the best treatment of DKA is prevention of DKA, by the adequate perioperative administration of insulin, and the assiduous avoidance of stress by the administration of adequate levels of anesthesia, use of regional anesthesia, careful avoidance of hypothermia, and excellent perioperative pain management.

The management of DKA is comprised of fluid/electrolyte therapy, and insulin therapy. Presumably, definitive DKA therapy will have been begun prior to the child's arrival in the operating room. However, urgent surgery for acute abdomens, for example, may not permit this to occur, and occasionally DKA will develop during surgery in a diabetic. It is therefore important for the anesthesiologist to know the principles of therapy.

#### ***O.R. Fluid therapy during DKA.***

The first priority is restoration of normal blood volume, which is generally accomplished by administration of normal saline or a balanced salt solution in quantities of 10-20 ml/kg, using the usual clinical signs to determine adequacy (skin turgor, urine output, pulse pressure and heart rate, etc.). It is very important to remember that seldom do children die because of dehydration and shock. The usual cause of death in children with DKA is idiopathic cerebral edema. This complication may be related to the quantity or rapidity of fluid therapy, although considerable controversy exists on this matter. Prudence therefore dictates cautious rates of rehydration. After restoration of adequate hemodynamics, the next priority is replacement of fluid deficits, a process that is generally accomplished using 1/2N saline with 40 mEq/L KCl, at a rate sufficient to replace the calculated deficit over 24-48 hrs. Ongoing intraoperative blood and fluid losses should be replaced as usually done with balanced salt solutions. Monitoring for the adequacy of fluid and electrolyte therapy must be an ongoing and frequent process, and frequent changes in the rate and/or composition of the fluid must be made as dictated by the behavior of the serum sodium, potassium, acid/base status, etc. In general, because of the concern of cerebral edema, it is better to err on the cautious side and give too little fluid than too much fluid.

#### ***O.R. Insulin Therapy during DKA***

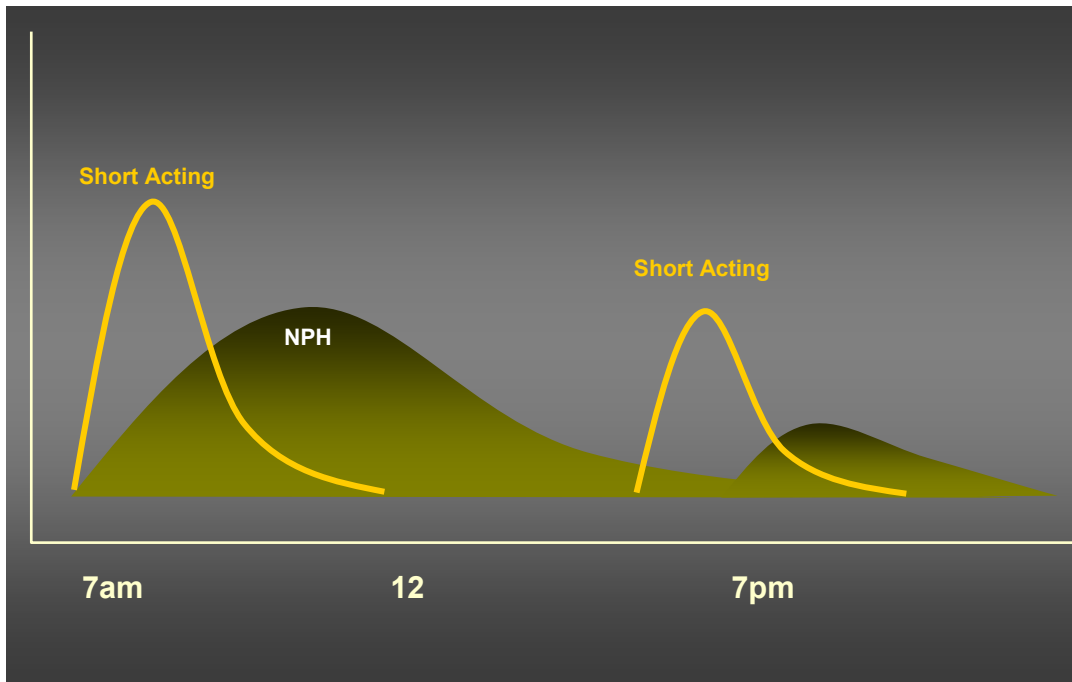
Physiologic insulin levels can be obtained by infusing insulin intravenously at a rate of 0.1 U/kg/hr. It is not necessary to bolus insulin prior to initiating an infusion. The insulin rate should be decreased when serum glucose has fallen to about 250 mg/dl unless the acidosis has not adequately been responding to therapy, in which case insulin resistance may exist. Insulin resistance may be caused by anti-insulin antibodies, or more frequently it may exist because of overactivity of counter-regulatory hormones excreted in response to the stress of illness and surgery. When the blood sugar has fallen to that level, supplemental glucose (100 mg/kg/hr) should be added to the intravenous infusions to prevent hypoglycemia. This is most easily done

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by converting the rehydration fluid to D51/2NS plus KCl. If insulin therapy continues at 0.1 U/kg/hr due to acidosis, then more glucose may be required to prevent hypoglycemia. In either case, glucose should continue to be monitored carefully and frequently in the operating room.

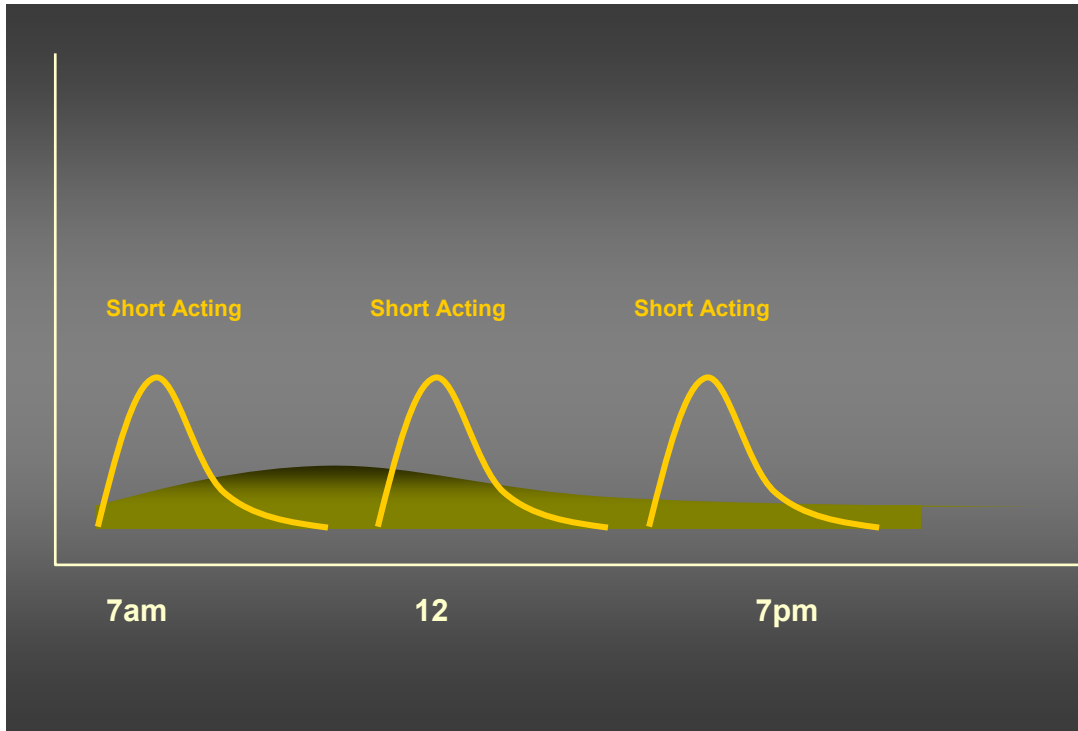
### Management of Diabetes in Elective Surgery

“Keep them sweet.” Most children with diabetes receive a combination of short and long acting insulin for day to day management of their disease. In past times, management would generally be with a once a day or twice a day injection of a combination of insulins (generally 2:1 NPH:regular), and conventional anesthesia teaching recommended giving diabetics have their long acting insulin as regular or CZI insulin the morning of surgery, and ignoring the short acting insulin they usually received. Further measurement of blood sugar then dictates further insulin management on a sliding scale, and moderate hyperglycemia was not only tolerated by the anesthesiologist, but desired. This traditional approach is no longer recommended.



**Figure 4.** Conventional daily insulin therapy using 2/3 NPH and 1/3 Regular combinations, given 2/3 in the morning and 1/3 at dinner. This approach is not usually used in children any longer.

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**Figure 5.** Current daily insulin therapy using a combination of ultra long acting preparations to provide basal insulin levels with 3 or 4 frequent small doses of short acting insulin to mimic the postprandial insulin levels in a normal subject.

More modern approaches to outpatient management of diabetic children attempt to produce more physiologic insulin levels sustained during the course of 24 hours, and greater euglycemia. It is now well recognized that “tight” control of JODM significantly reduces diabetic end organ complications in later life.

Clearly, the old approach of  $\frac{1}{2}$  the quantity of long acting insulin and none of the short acting insulin will significantly under dose the child on this regimen, and new strategies must be used.

Continuous insulin therapy has the advantages of being more flexible and titratable than intermittent s.c. insulin, it is associated with less hypo- and hyperglycemia, and is probably therefore associated with a lower incidence of wound infection, faster healing, and improved patient outcome.

Appropriate techniques are:

1. Withhold the usual a.m. dose of insulin.
2. When patient arrives in hospital, begin an infusion with glucose at maintenance glucose rates (1.5 mg/kg/min or 100 mg/kg/hr, e.g. D51/2NS + 20 KCl/L at 2 ml/kg/hr).
3. Start a continuous insulin infusion at:
  - a. 0.05 U/kg/hr.
  - b. or, the total daily insulin dose usually used  $\div$  24.
  - c. or, at 1U for every 2 grams of glucose infused intravenously (1:1 if CPB).
  - d. or, according to a sliding scale, units/kg/hr = serum glucose/3500.
  - e. Follow serum glucose q1hr and adjust insulin accordingly.

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### Summary

1. The mortality associated with JODM is primarily due to DKA
2. The mortality associated with DKA is primarily due to cerebral edema
3. During DKA, it is easy to underestimate the magnitude of fluid losses
4. During DKA, the serum sodium will overestimate deficits
5. During DKA, the serum potassium will underestimate deficits
6. When serum glucose >300 mg/dl, the serum glucose is more affected by renal function than by insulin.
7. The adequacy of insulin therapy is best judged by the resolution of acidosis, not hyperglycemia.
8. Tight control of blood sugar in the nonketoacidotic diabetic is associated with better patient outcome.

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