

Diabetes Mellitus

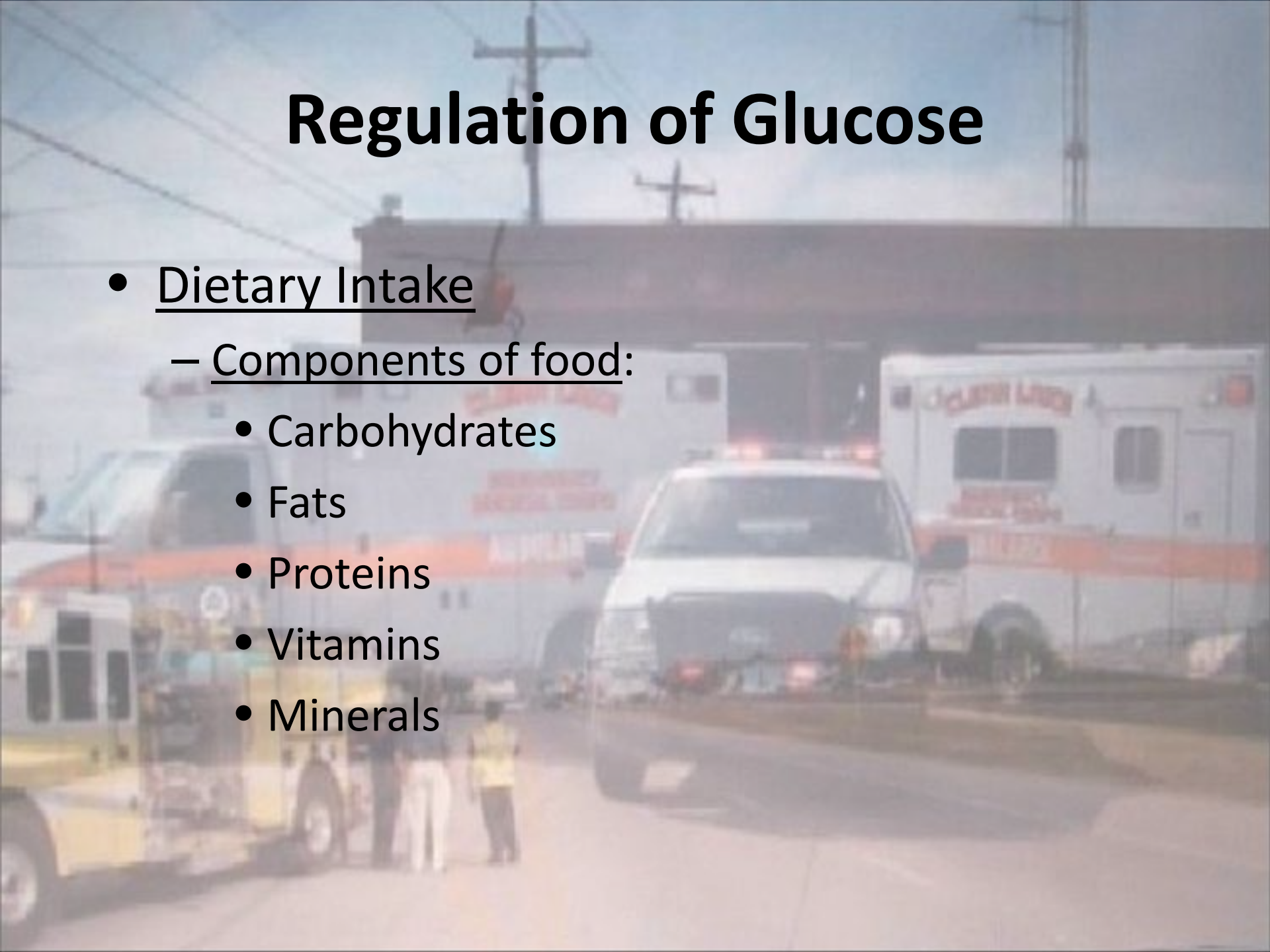


Diabetes Mellitus

- Chronic metabolic disease
- One of the most common diseases in North America
 - Affects 5% of USA population (12 million people)
- Results in
 - ↓ insulin secretion by the Beta (β) cells of the islets of Langerhans in the pancreas, AND/OR
 - Defects in insulin receptors on cell membranes leading to cellular resistance to insulin
- Leads to an ↑ risk for significant cardiovascular, renal and ophthalmic disease

Regulation of Glucose

- Dietary Intake
 - Components of food:
 - Carbohydrates
 - Fats
 - Proteins
 - Vitamins
 - Minerals



Regulation of Glucose

- The other 3 major food sources for glucose are
 - carbohydrates
 - proteins
 - fats
- Most sugars in the human diet are complex and must be broken down into simple sugars: glucose, galactose and fructose - before use

Regulation of Glucose

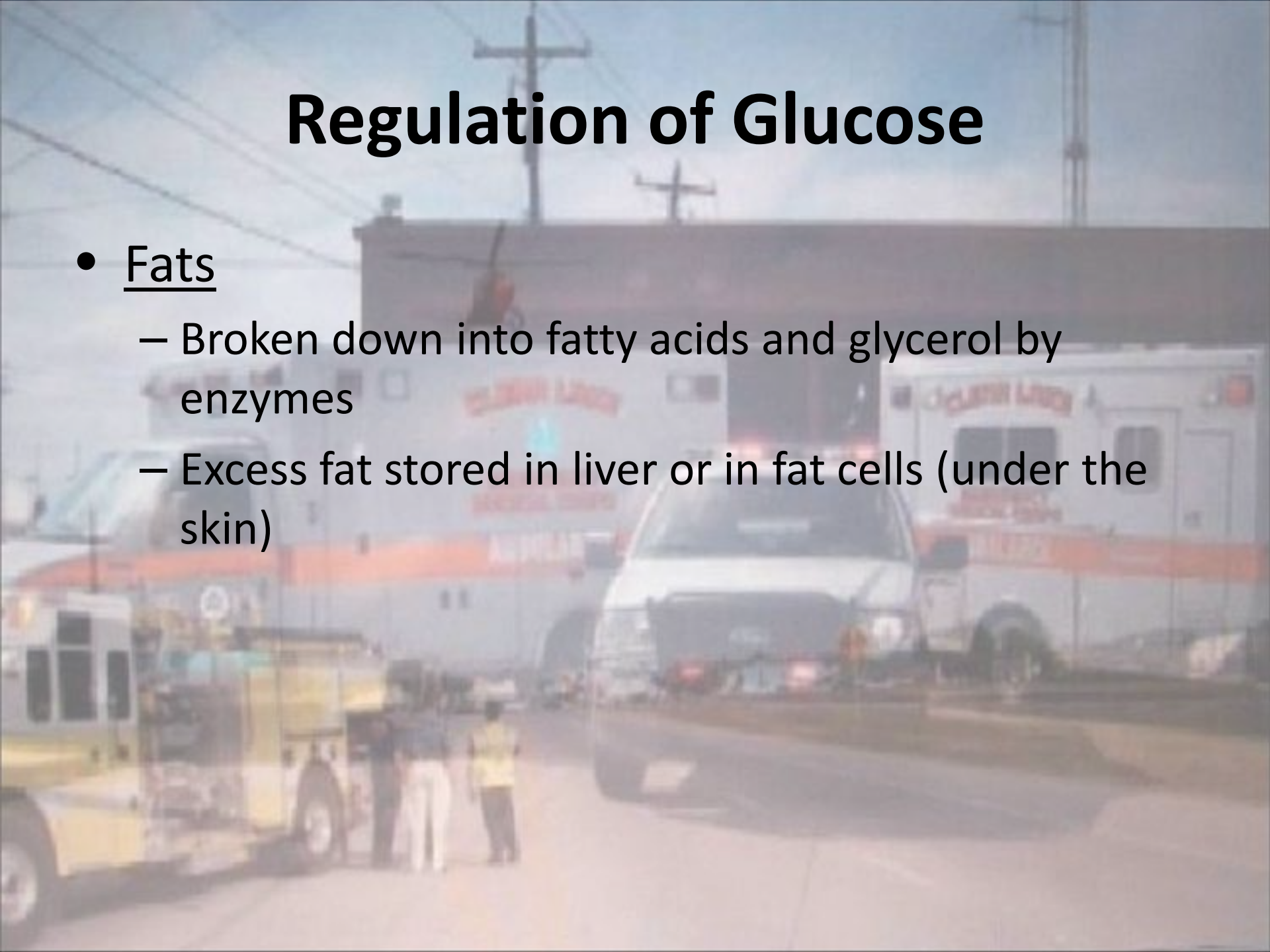
- Carbohydrates

- Found in sugary, starchy foods
- Ready source of near-instant energy
- If not “burned” immediately by body, stored in liver and skeletal muscle as glycogen (short-term energy) or as fat (long-term energy needs)
- After normal meal, approximately 60% of the glucose is stored in liver as glycogen

Regulation of Glucose

- Fats

- Broken down into fatty acids and glycerol by enzymes
- Excess fat stored in liver or in fat cells (under the skin)



Regulation of Glucose

- Pancreatic hormones are required to regulate blood glucose level
 - *glucagon* released by Alpha (α) cells
 - *insulin* released by Beta Cells (β)
 - *somatostatin* released by Delta Cells (δ)

Regulation of Glucose

- Alpha (α) cells release *glucagon* to control blood glucose level
 - When blood glucose levels fall, α cells \uparrow the amount of glucagon in the blood
 - The surge of glucagon stimulates liver to release glucose stores by the breakdown of glycogen into glucose (glycogenolysis)
 - Also, glucagon stimulates the liver to produce glucose (gluconeogenesis)

Regulation of Glucose

- Beta Cells (β) release *insulin* (antagonistic to glucagon) to control blood glucose level
 - Insulin \uparrow the rate at which various body cells take up glucose \Rightarrow insulin lowers the blood glucose level
 - Promotes glycogenesis - storage of glycogen in the liver
 - Insulin is rapidly broken down by the liver and must be secreted constantly

Regulation of Glucose

- Delta Cells (δ) produce *somatostatin*, which inhibits both glucagon and insulin
 - inhibits insulin and glucagon secretion by the pancreas
 - inhibits digestion by inhibiting secretion of digestive enzymes
 - inhibits gastric motility
 - inhibits absorption of glucose in the intestine

Regulation of Glucose

- Breakdown of sugars carried out by enzymes in the GI system
 - As simple sugars, they are absorbed from the GI system into the body
- To be converted into energy, glucose must first be transmitted through the cell membrane
 - Glucose molecule is too large and does not readily diffuse

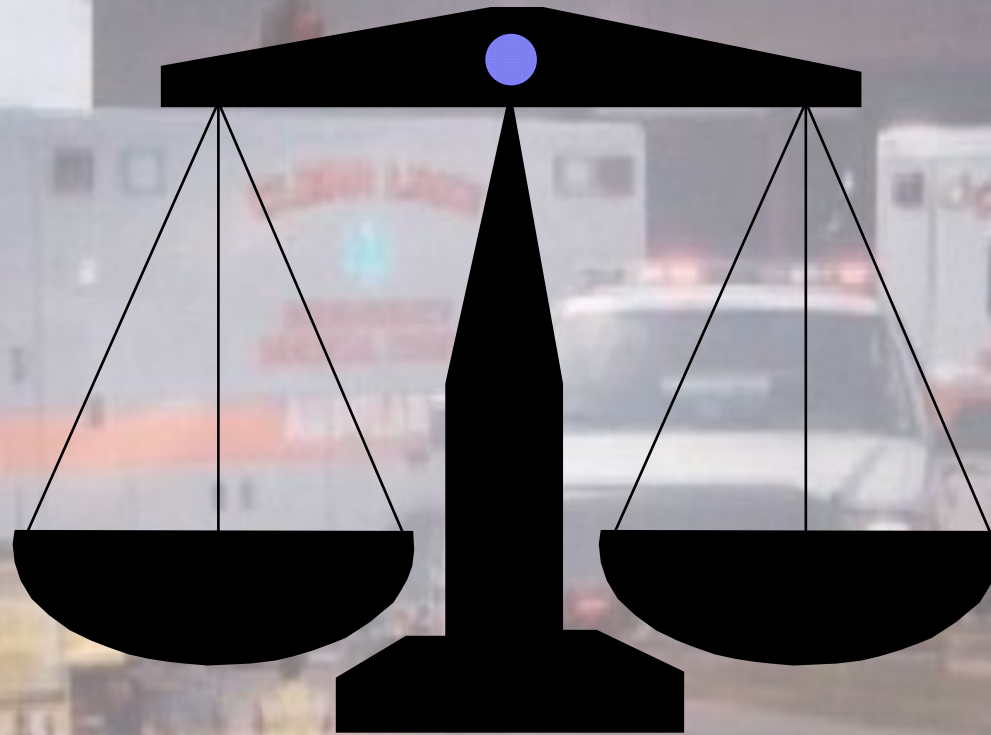
Regulation of Glucose

- Glucose must pass into the cell by binding to a special carrier protein on the cell's surface.
 - *Facilitated diffusion* - carrier protein binds with the glucose and carries it into the cell.
- The rate at which glucose can enter the cell is dependent upon insulin levels
 - Insulin serves as the messenger - travels via blood to target tissues
 - Combines with specific insulin receptors on the surface of the cell membrane

Regulation of Glucose

- Body strives to maintain blood glucose between 60 mg/dl and 120 mg/dl.
- Glucose
 - brain is the biggest user of glucose in the body
 - sole energy source for brain
 - brain does not require insulin to utilize glucose

Regulation of Glucose



Glucagon and Insulin are opposites (antagonists) of each other.

Regulation of Glucose

- Glucagon
 - Released in response to:
 - Sympathetic stimulation
 - Decreasing blood glucose concentration
 - Acts primarily on liver to increase rate of glycogen breakdown
 - Increasing blood glucose levels have inhibitory effect on glucagon secretion

Regulation of Glucose

- Insulin

- Released in response to:

- Increasing blood glucose concentration
- Parasympathetic innervation

- Acts on cell membranes to increase glucose uptake from blood stream

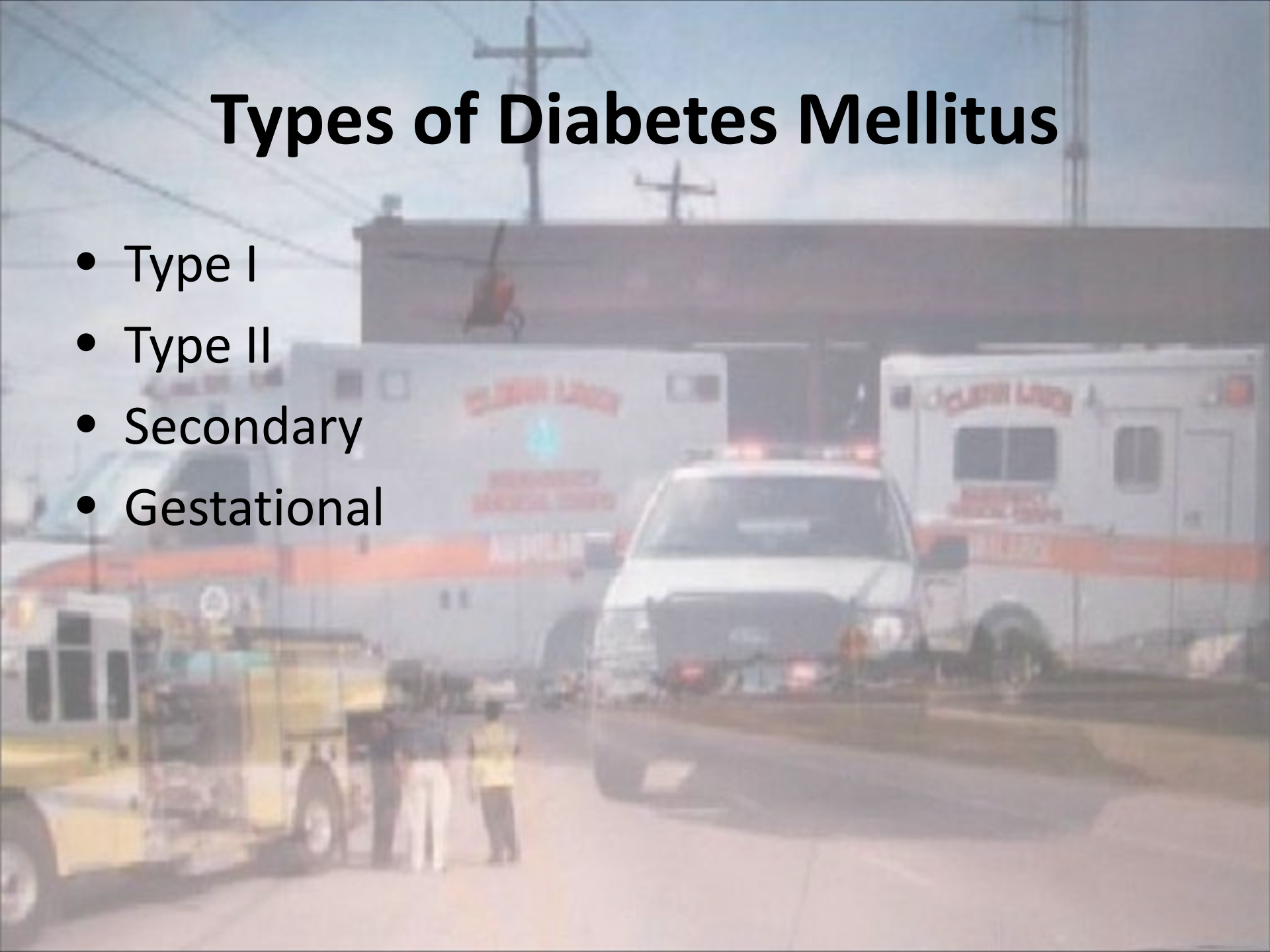
- Promotes facilitated diffusion of glucose into cells

Diabetes Mellitus

- 2 Types historically based on age of onset (NOT insulin vs. non-insulin)
 - Type I
 - juvenile onset
 - insulin dependent
 - Type II
 - historically adult onset
 - now some morbidly obese children are developing Type II diabetes
 - non-insulin dependent
 - may progress to insulin dependency

Types of Diabetes Mellitus

- Type I
- Type II
- Secondary
- Gestational



Pathophysiology of Type I Diabetes Mellitus

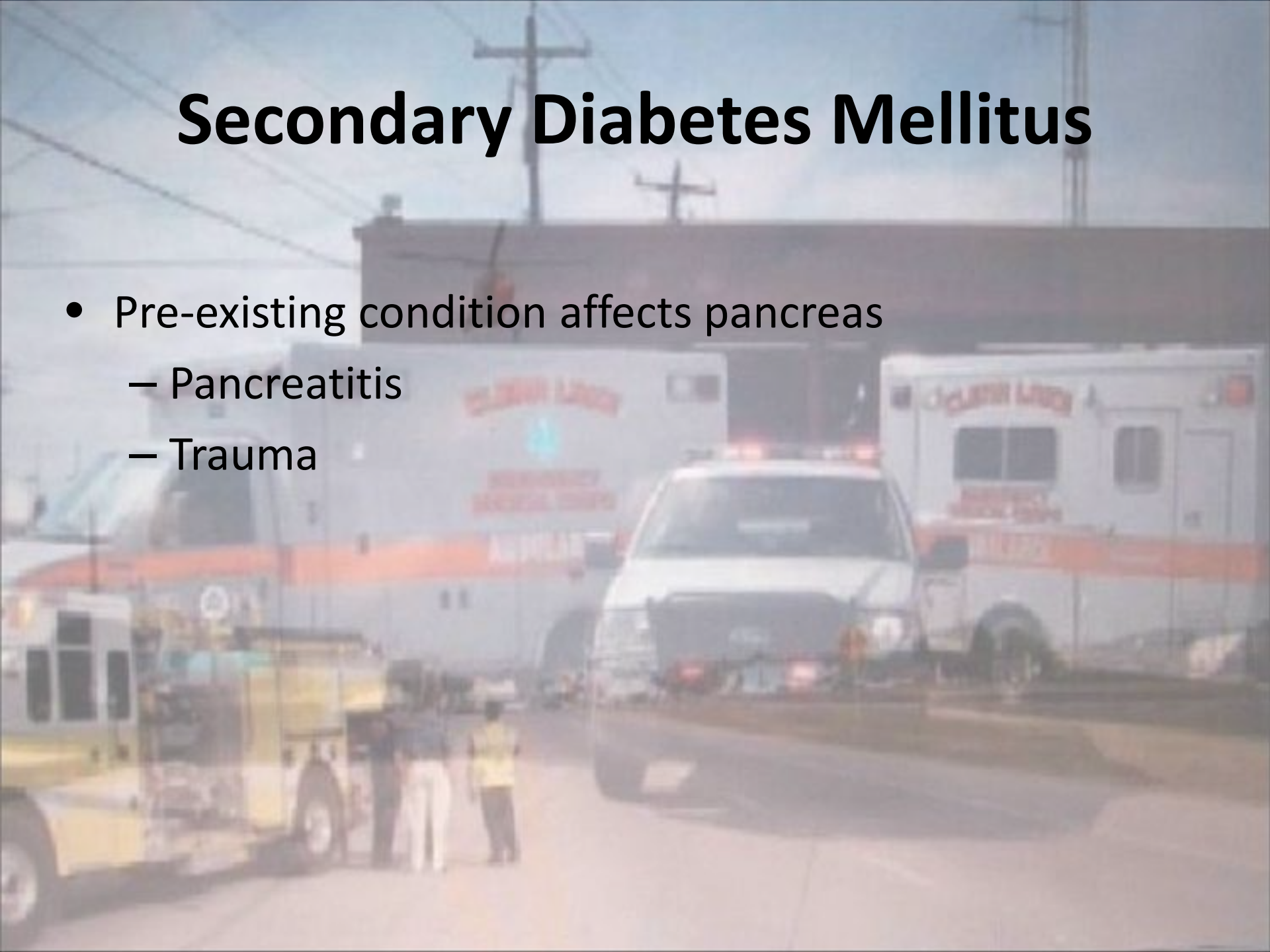
- Characterized by inadequate or absent production of insulin by pancreas
- Usually presents by age 25
- Strong genetic component
- Autoimmune features
 - body destroys own insulin-producing cells in pancreas
 - may follow severe viral illness or injury
- Requires lifelong treatment with insulin replacement

Pathophysiology of Type II Diabetes Mellitus

- Pancreas continues to produce some insulin however disease results from combination of:
 - Relative insulin deficiency
 - Decreased sensitivity of insulin receptors
- Onset usually after age 25 in overweight adults
 - Some morbidly obese children develop Type II diabetes
- Familial component
- Usually controlled with diet, weight loss, oral hypoglycemic agents
 - Insulin may be needed at some point in life

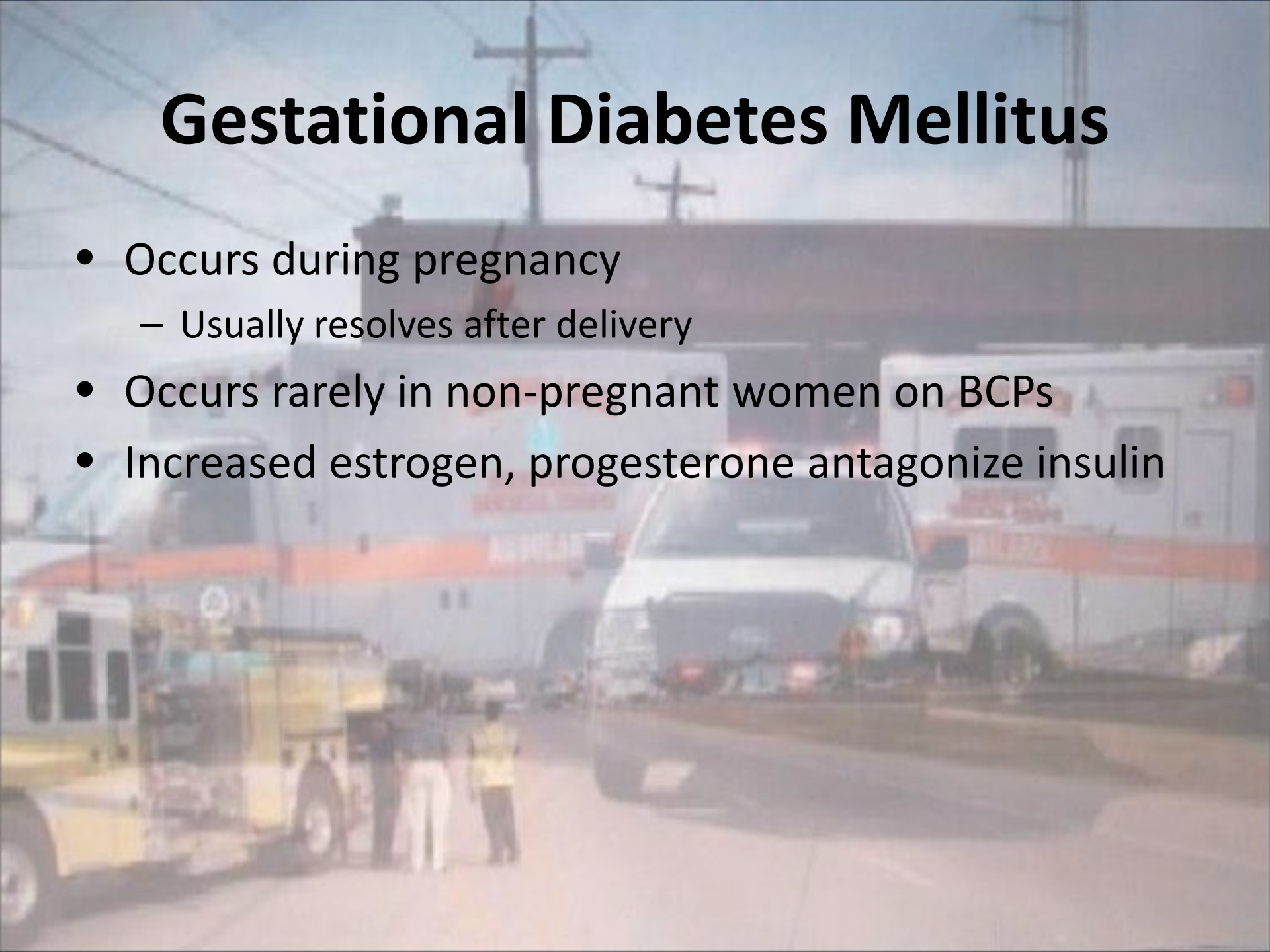
Secondary Diabetes Mellitus

- Pre-existing condition affects pancreas
 - Pancreatitis
 - Trauma



Gestational Diabetes Mellitus

- Occurs during pregnancy
 - Usually resolves after delivery
- Occurs rarely in non-pregnant women on BCPs
- Increased estrogen, progesterone antagonize insulin



Presentation of New Onset Diabetes Mellitus

- 3 Ps
 - Polyuria
 - Polydipsia
 - Polyphagia
- Blurred vision, dizziness, altered mental status
- Rapid weight loss
- Warm dry skin,
- Weakness, Tachycardia, Dehydration

Long Term Treatment of Diabetes Mellitus

- Diet regulation
 - *e.g.* 1400 calorie ADA diet
- Exercise
 - increase patient's glucose metabolism
- Oral hypoglycemic agents
 - Sulfonylureas
- Insulin
 - Historically produced from pigs (porcine insulin)
 - Currently genetic engineering has lead to human insulin (Humulin)

Long Term Treatment of Diabetes Mellitus

- Insulin

- Available in various forms distinguished on onset and duration of action

- Onset

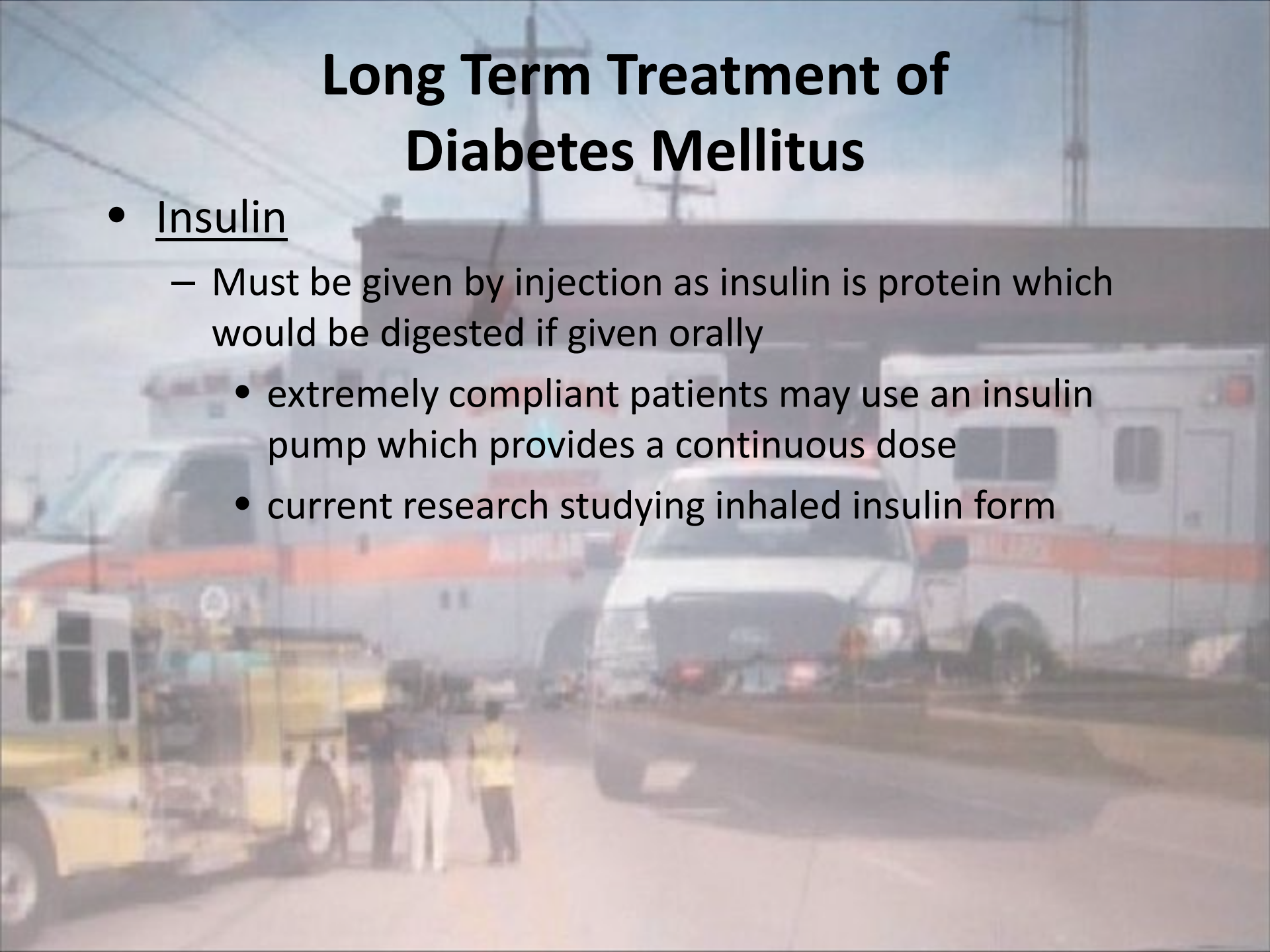
- rapid (Regular, Semilente, Novolin 70/30)
 - intermediate (Novolin N, Lente)
 - slow (Ultralente)

- Duration

- short, 5-7 hrs (Regular)
 - intermediate, 18-24 hrs (Semilente, Novolin N, Lente, NPH)
 - long-acting, 24 - 36+ hrs (Novolin 70/30, Ultralente)

Long Term Treatment of Diabetes Mellitus

- Insulin
 - Must be given by injection as insulin is protein which would be digested if given orally
 - extremely compliant patients may use an insulin pump which provides a continuous dose
 - current research studying inhaled insulin form

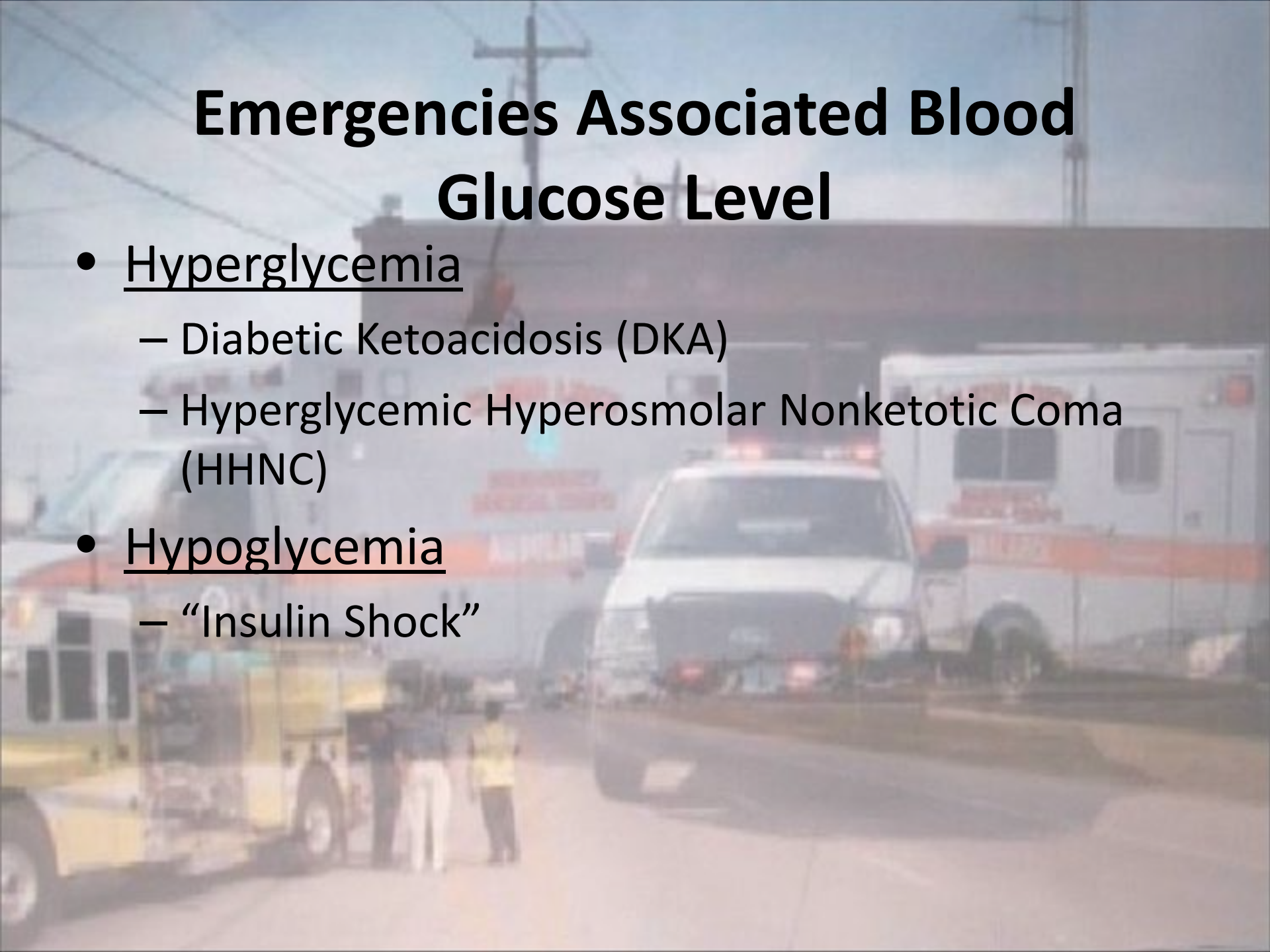


Long Term Treatment of Diabetes Mellitus

- Oral Hypoglycemic Agents
 - Stimulate the release of insulin from the pancreas, thus patient must still have intact *beta* cells in the pancreas.
 - Common agents include:
 - Glucotrol[®] (glipizide)
 - Micronase[®] or Diabeta[®] (glyburide)
 - Glucophage[®] (metformin) [Not a sulfonylurea]

Emergencies Associated Blood Glucose Level

- Hyperglycemia
 - Diabetic Ketoacidosis (DKA)
 - Hyperglycemic Hyperosmolar Nonketotic Coma (HHNC)
- Hypoglycemia
 - “Insulin Shock”

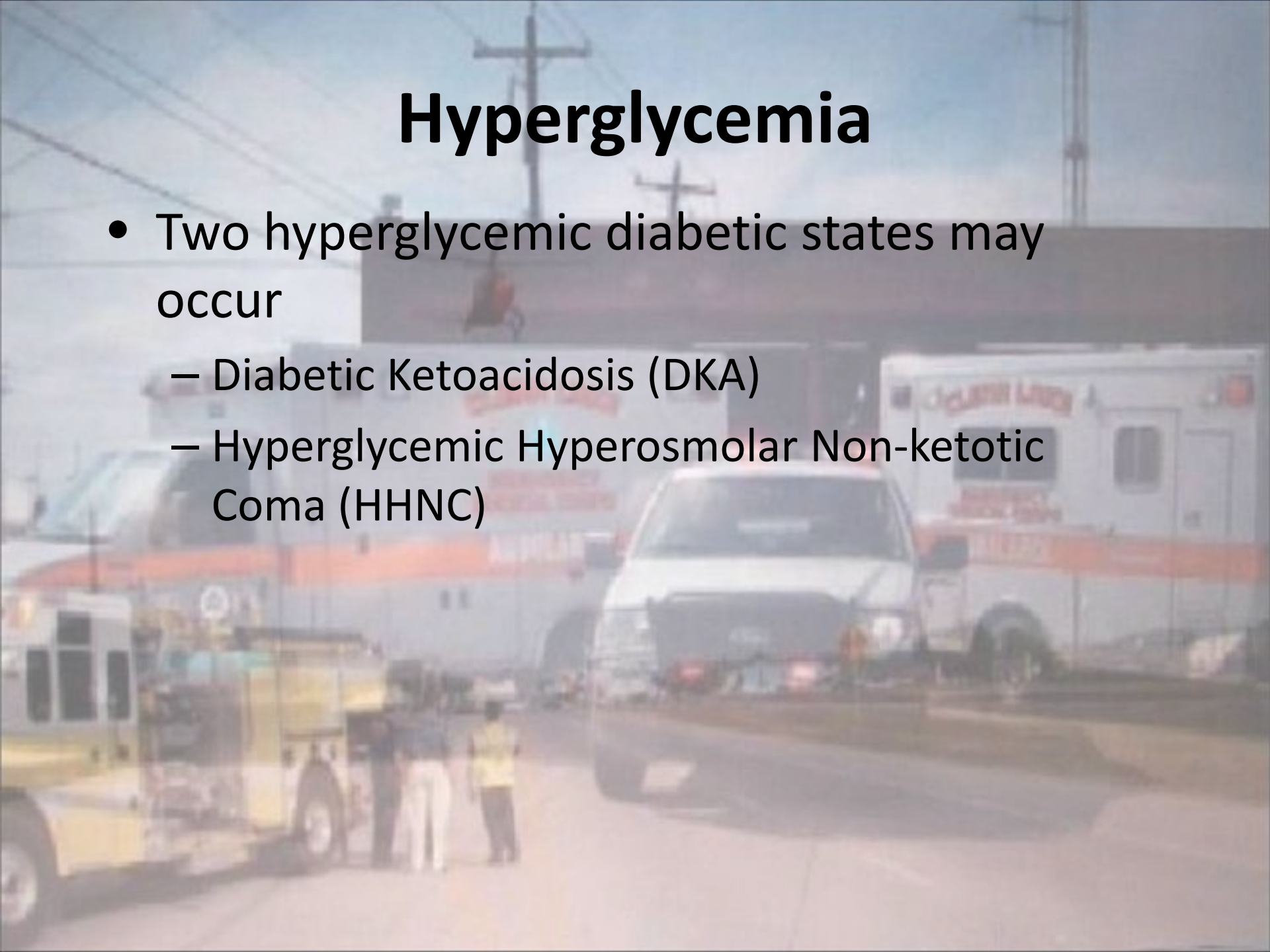


Hyperglycemia

- Defined as blood glucose > 200 mg/dl
- Causes
 - Failure to take *medication* (insulin)
 - Increased *dietary* intake
 - *Stress* (surgery, MI, CVA, trauma)
 - Fever
 - *Infection*
 - *Pregnancy* (gestational diabetes)

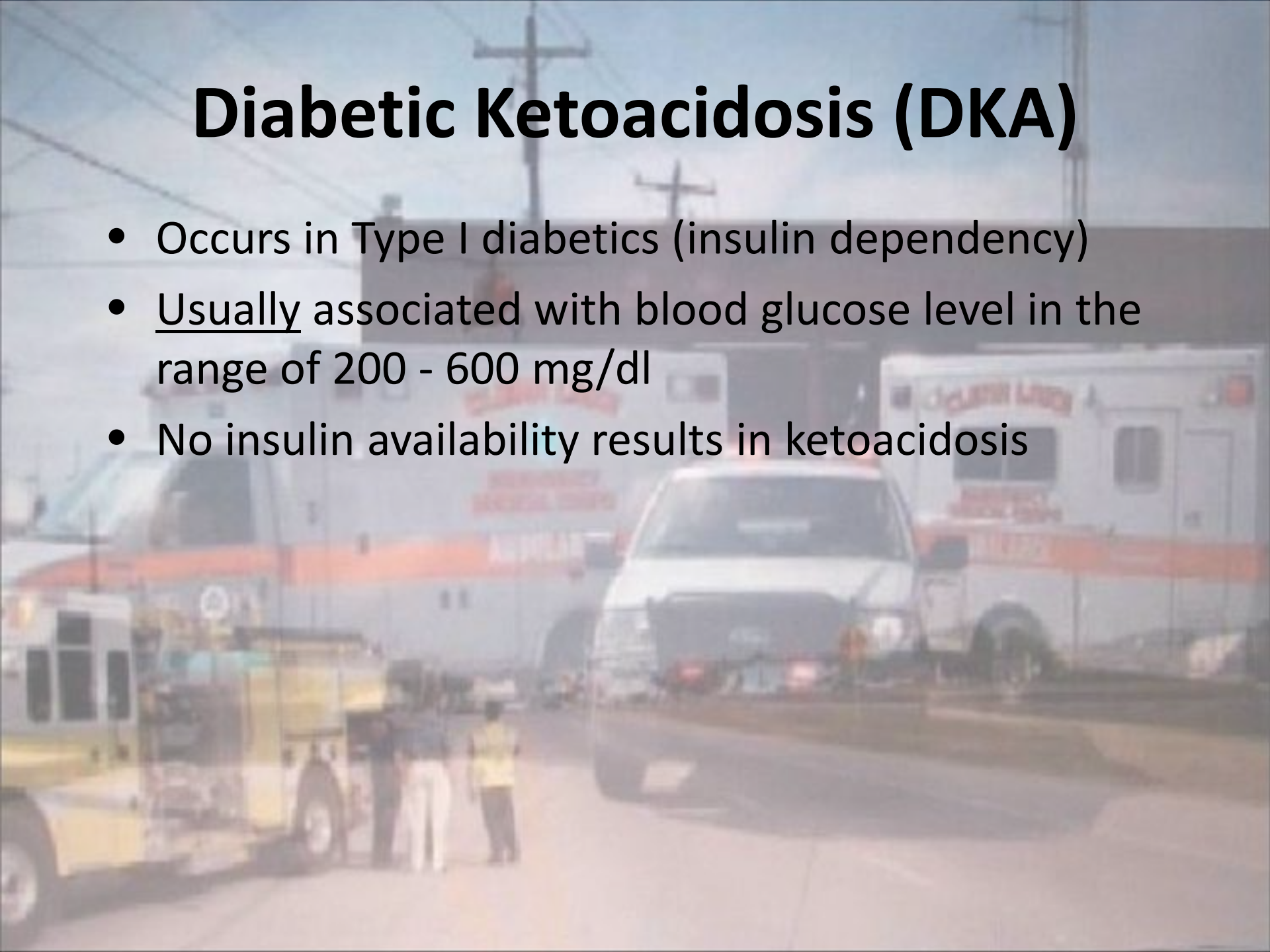
Hyperglycemia

- Two hyperglycemic diabetic states may occur
 - Diabetic Ketoacidosis (DKA)
 - Hyperglycemic Hyperosmolar Non-ketotic Coma (HHNC)



Diabetic Ketoacidosis (DKA)

- Occurs in Type I diabetics (insulin dependency)
- Usually associated with blood glucose level in the range of 200 - 600 mg/dl
- No insulin availability results in ketoacidosis



Diabetic Ketoacidosis (DKA)

- Pathophysiology
 - Results from absence of insulin
 - prevents glucose from entering the cells
 - leads to glucose accumulation in the blood
 - Cells become starved for glucose and begin to use other energy sources (primarily fats)
 - Fat metabolism generates fatty acids
 - Further metabolized into ketoacids (ketone bodies)

Diabetic Ketoacidosis (DKA)

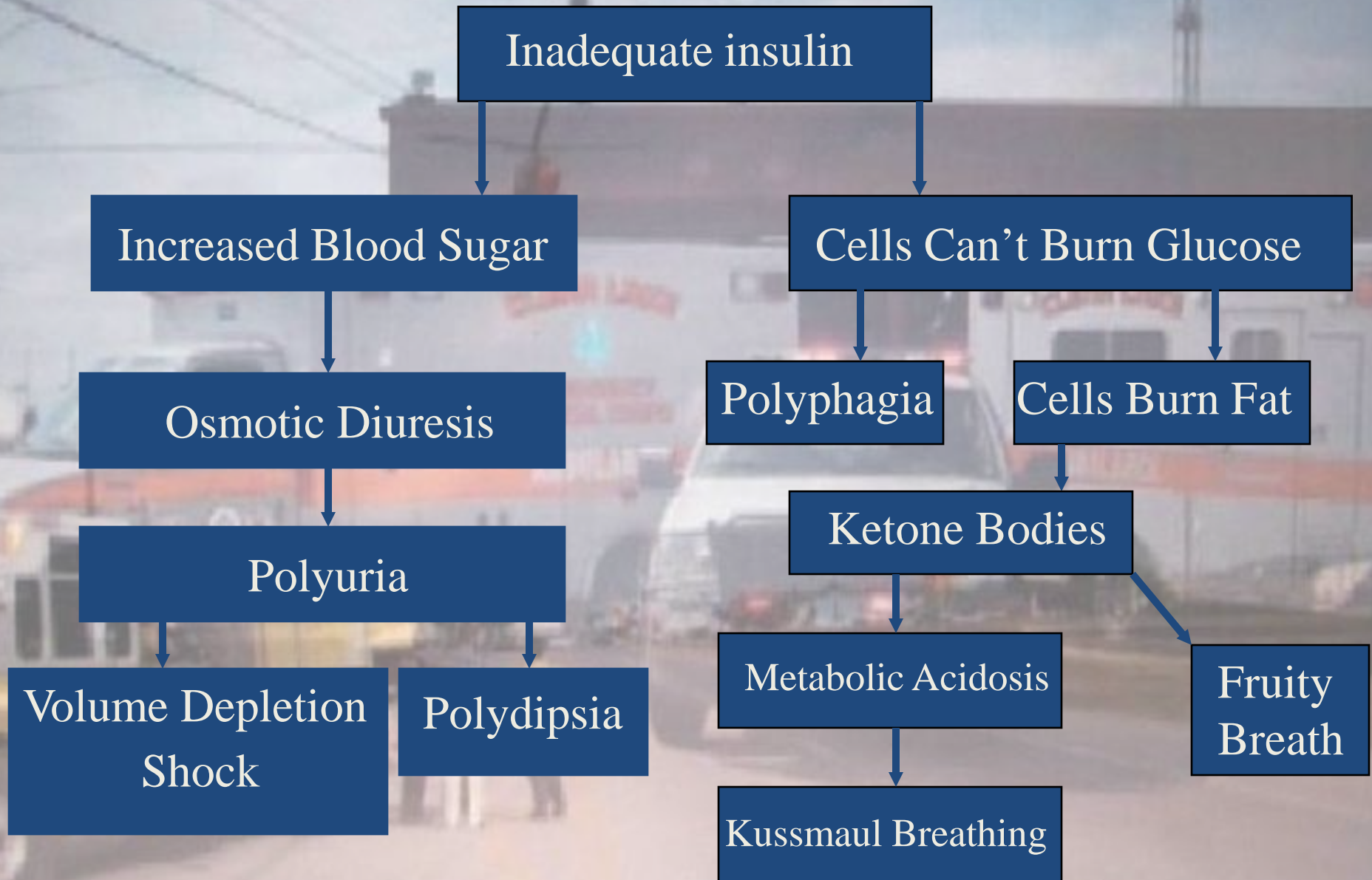
- Pathophysiology (cont)
 - Blood sugar rises above renal threshold for reabsorption (blood glucose > 180 mg/dl)
 - glucose “spills” into the urine
 - Loss of glucose in urine causes osmotic diuresis
 - Results in
 - dehydration
 - acidosis
 - electrolyte imbalances (especially K^+)

Diabetic Ketoacidosis (DKA)

- Presentation

- Gradual onset with progression
- Warm, pink, dry skin
- Dry mucous membranes (dehydrated)
- Tachycardia, weak peripheral pulses
- Weight loss
- Polyuria (frequent urination)
- Polydipsia (excessive thirst)
- Abdominal pain with nausea/vomiting
- Altered mental status
- Kussmaul respirations with acetone (fruity) odor

Diabetic Ketoacidosis



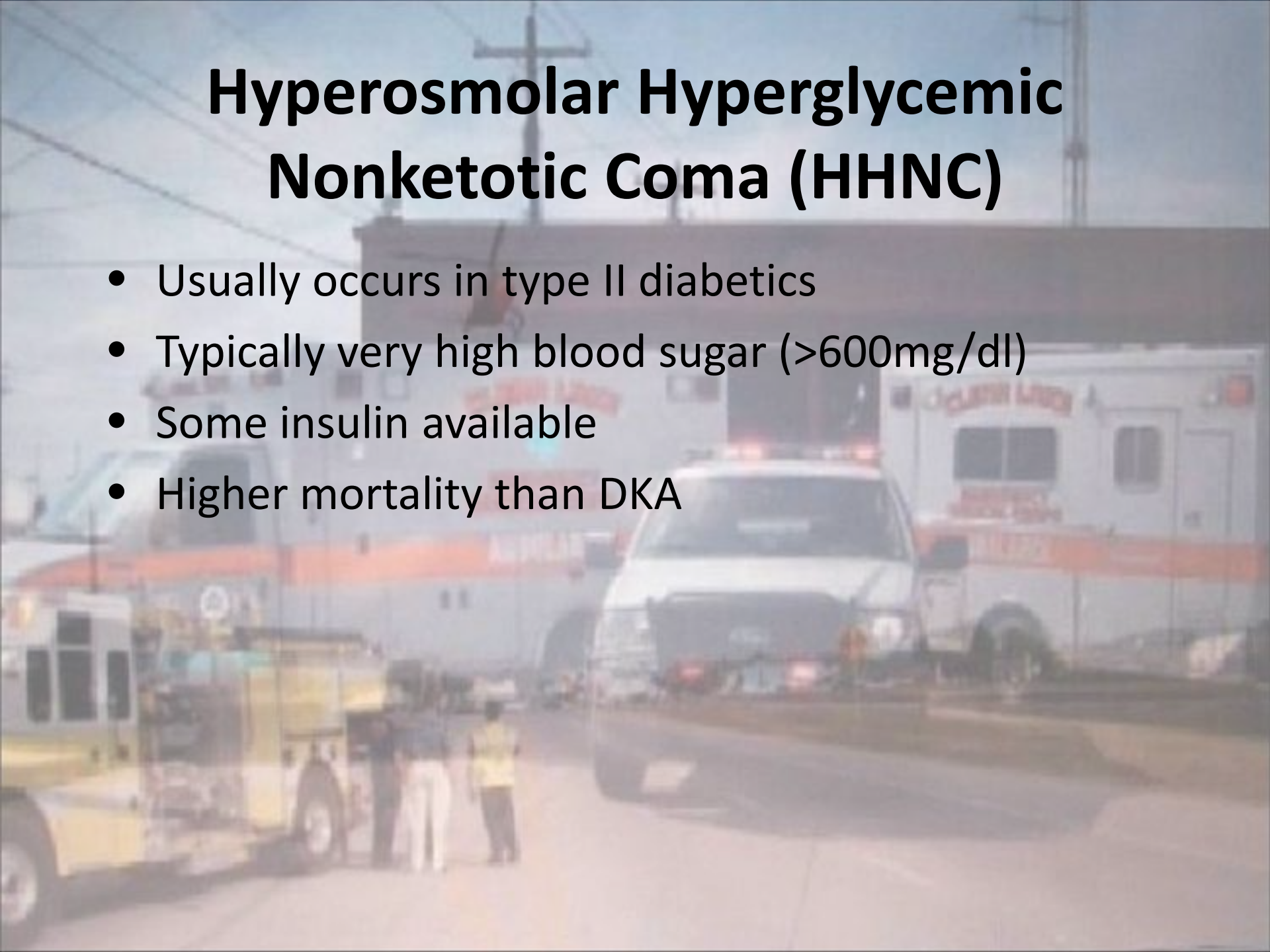
Management of DKA

- Airway/Ventilation/Oxygen NRB mask
- Assess blood glucose level & ECG
- IV access, large bore NS
 - normal saline bolus and reassess
 - often requires several liters
- Assess for underlying cause of DKA
- Transport

How does fluid treat DKA?

Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

- Usually occurs in type II diabetics
- Typically very high blood sugar (>600mg/dl)
- Some insulin available
- Higher mortality than DKA



Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

- Pathophysiology
 - Some minimal insulin production
 - enough insulin available to allow glucose to enter the cells and prevent ketogenesis
 - not enough to decrease gluconeogenesis by liver
 - no ketosis
 - Extreme hyperglycemia produces hyperosmolar state causing
 - diuresis
 - severe dehydration
 - electrolyte disturbances

Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

Inadequate insulin

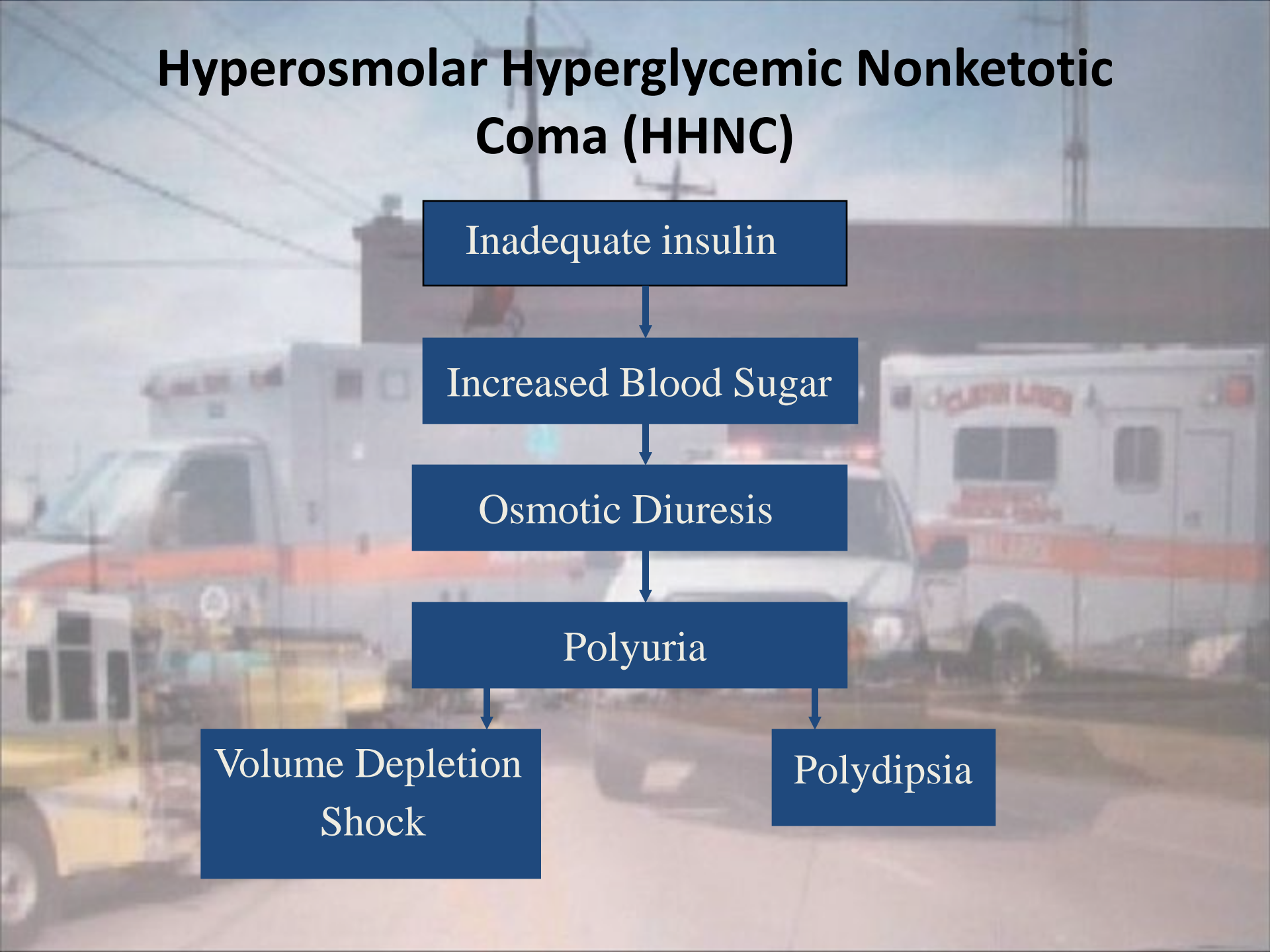
Increased Blood Sugar

Osmotic Diuresis

Polyuria

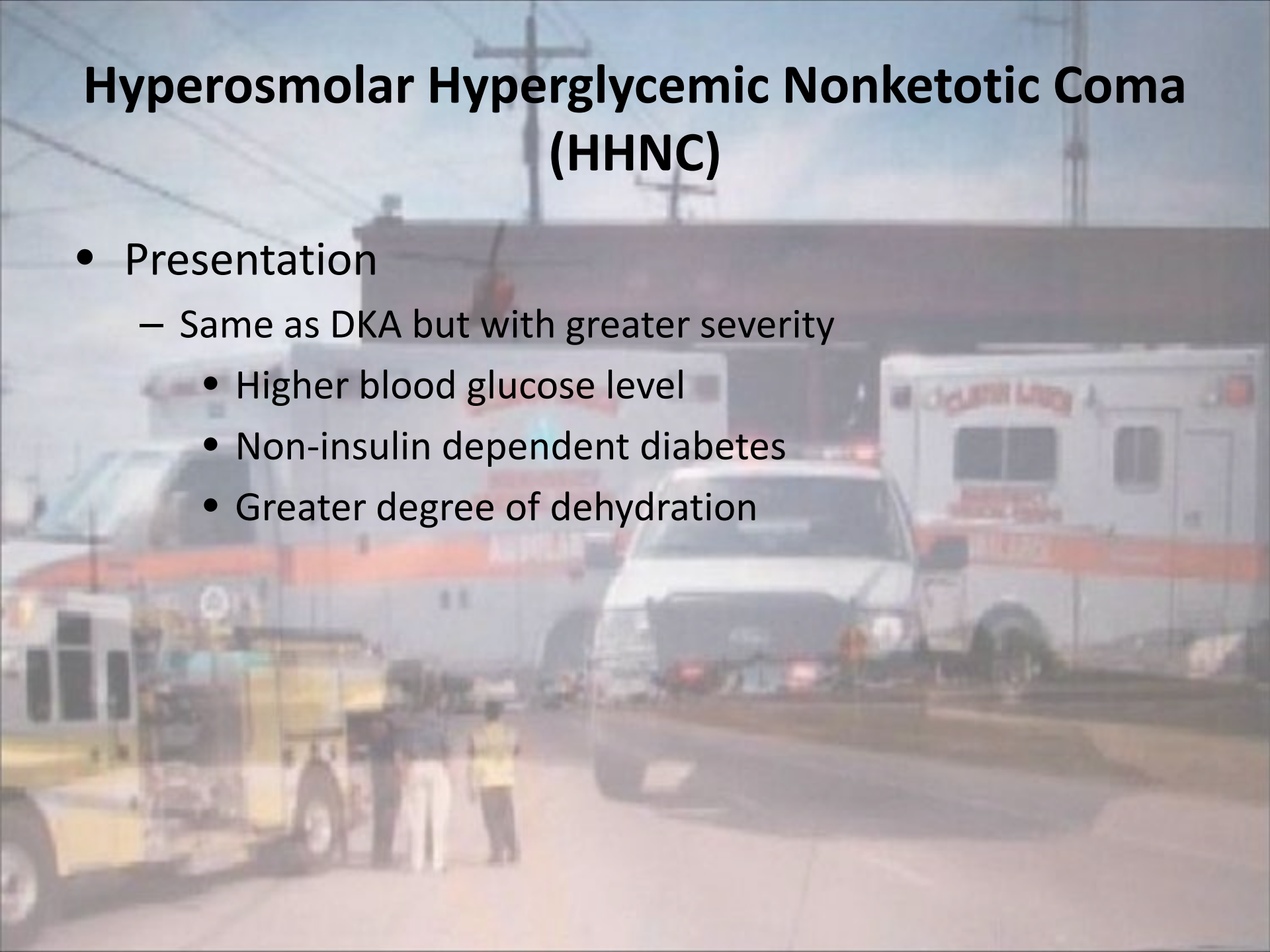
Volume Depletion
Shock

Polydipsia



Hyperosmolar Hyperglycemic Nonketotic Coma (HHNC)

- Presentation
 - Same as DKA but with greater severity
 - Higher blood glucose level
 - Non-insulin dependent diabetes
 - Greater degree of dehydration



Management of HHNC

- Secure airway and assess ventilation
 - Consider need to assist ventilation
 - Consider need to intubate
- High concentration oxygen
- Assess blood glucose level & ECG
- IV access, large bore NS
 - normal saline bolus and reassess
 - often requires several liters
- Assess for underlying cause of HHNC
- Transport

Further Management of Hyperglycemia

- Insulin (regular)
 - Correct hyperglycemia
- Correction of acid/base imbalances
 - Bicarbonate (severe cases documented by ABG)
- Normalization of electrolyte balance
 - DKA may result in hyperkalemia 2° to acidosis
 - H^+ shifts intracellularly, K^+ moves to extracellular space
 - Urinary K^+ losses may lead to hypokalemia once therapy is started

Hypoglycemia

- True hypoglycemia defined as blood sugar < 60 mg/dl
- ALL hypoglycemia is NOT caused by diabetes
 - Can occur in non-diabetic patients
 - thin young females
 - alcoholics with liver disease
 - alcohol consumption on empty stomach will block glucose synthesis in liver (gluconeogenesis)
- Hypoglycemia causes impaired functioning of brain which relies on constant supply of glucose

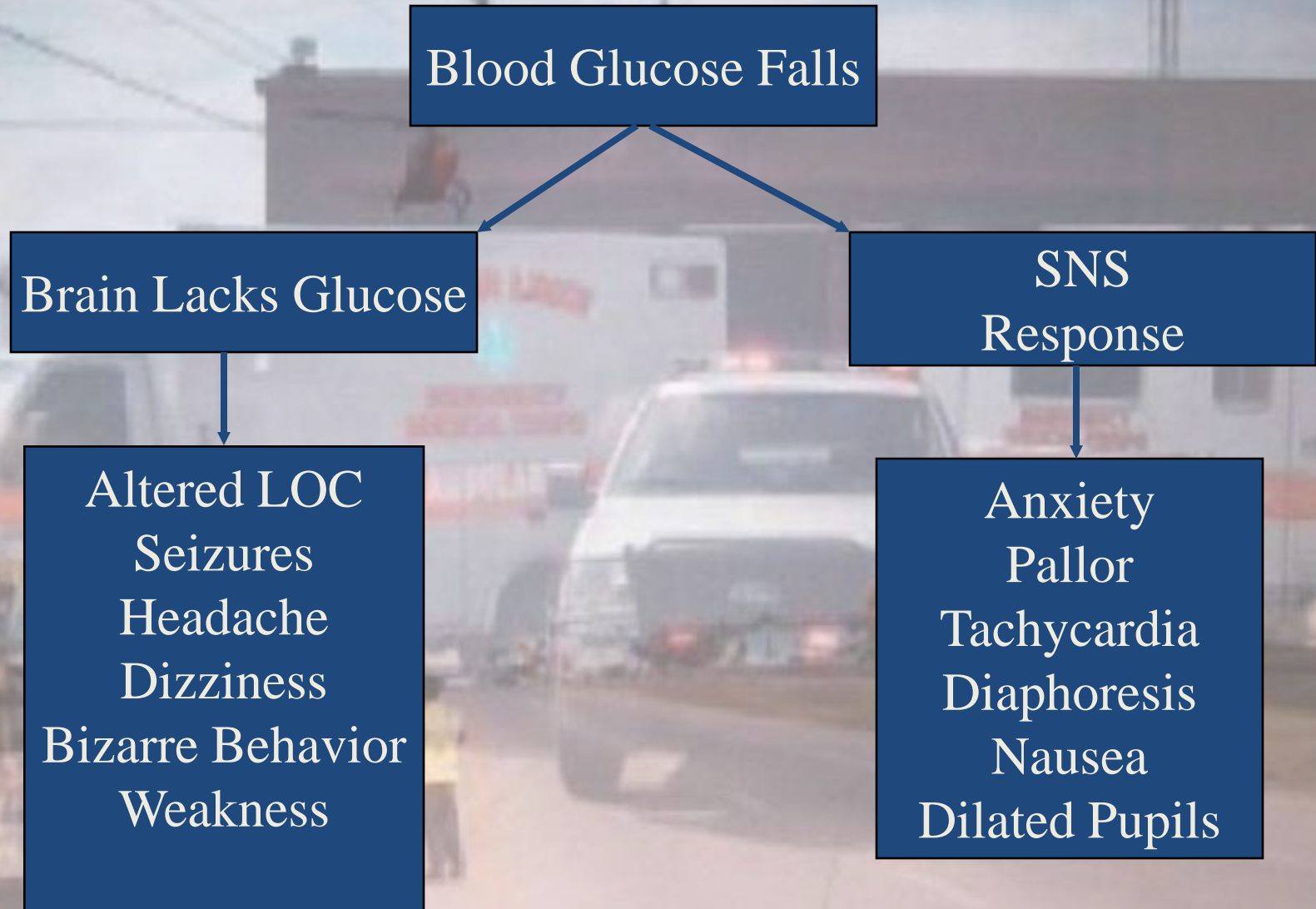
Hypoglycemia

- Causes of hypoglycemia in diabetics
 - Too much insulin
 - Too much oral hypoglycemic agent
 - Long half-life requires hospitalization
 - Decreased dietary intake (took insulin and missed meal)
 - Vigorous physical activity
- Pathophysiology
 - Inadequate blood glucose available to brain and other cells resulting from one of the above causes

Hypoglycemia

- Presentation
 - Hunger (initially), Headache
 - Weakness, Incoordination (*mimics a stroke*)
 - Confusion, Unusual behavior
 - may appear intoxicated
 - Seizures
 - Coma
 - Weak, rapid pulse
 - Cold, clammy skin
 - Nervousness, trembling, irritability

Hypoglycemia: Pathophysiology



Hypoglycemia

Beta Blockers may mask symptoms by inhibiting sympathetic response

Management of Hypoglycemia

- Secure airway manually
 - suction prn
 - Ventilate prn
- High concentration oxygen
- Vascular access
 - Large bore IV catheter
 - Saline lock, D₅W or NS
 - Large proximal vein preferred
- Assess blood glucose level

Management of Hypoglycemia

- Oral glucose
 - ONLY if intact gag reflex, awake & able to sit up
 - 15gm-30gm of packaged glucose, or
 - May use sugar-containing drink or food
 - Oral route often slower
- Intravenous glucose
 - Adult: Dextrose 50% (D₅₀) 25gms IV in patent, free-flowing vein, may repeat
 - Children: Dextrose 25% (D₂₅) @ 2 - 4 cc/kg (0.5 - 1 gm/kg)
[Infants - may choose Dextrose 10% @ 0.5 - 1 gm/kg or 5 - 10 cc/kg]

Management of Hypoglycemia

- Glucagon
 - Used if unable to obtain IV access
 - 1 mg IM
 - Requires glycogen stores
 - slower onset of action than IV route

What persons are likely to have inadequate glycogen stores?

Management of Hypoglycemia

- Have patient eat high-carbohydrate meal
- Transport?
 - Patient Refusal
 - Leave only with responsible family/friend for 6 hours
 - Must educate family/friend to hypoglycemic signs/symptoms
 - Advise to contact personal physician
 - Transport
 - Hypoglycemic patients on oral agents (long half life)
 - Unknown, atypical or untreated cause of hypoglycemia

Long-term Complications of Diabetes Mellitus

- Blindness
 - Retinal hemorrhages
- Renal Disease
- Peripheral Neuropathy
 - Numbness in “stocking glove” distribution (hands and feet)
- Heart Disease and Stroke
 - Chronic state of Hyperglycemia leads to early atherosclerosis
- Complications in Pregnancy

Long-term Complications of Diabetes Mellitus

- Diffuse Atherosclerosis
 - AMI
 - CVA
 - PVD
 - Hypertension
 - Renal failure
 - Diabetic retinopathy/blindness
 - Gangrene



Long-term Complications of Diabetes Mellitus

Diabetics are up to 4 times more likely to have heart disease and up to 6 times more likely to have a stroke than a non-diabetic

10% of all diabetics develop renal disease usually resulting in dialysis

Long-term Complications of Diabetes Mellitus

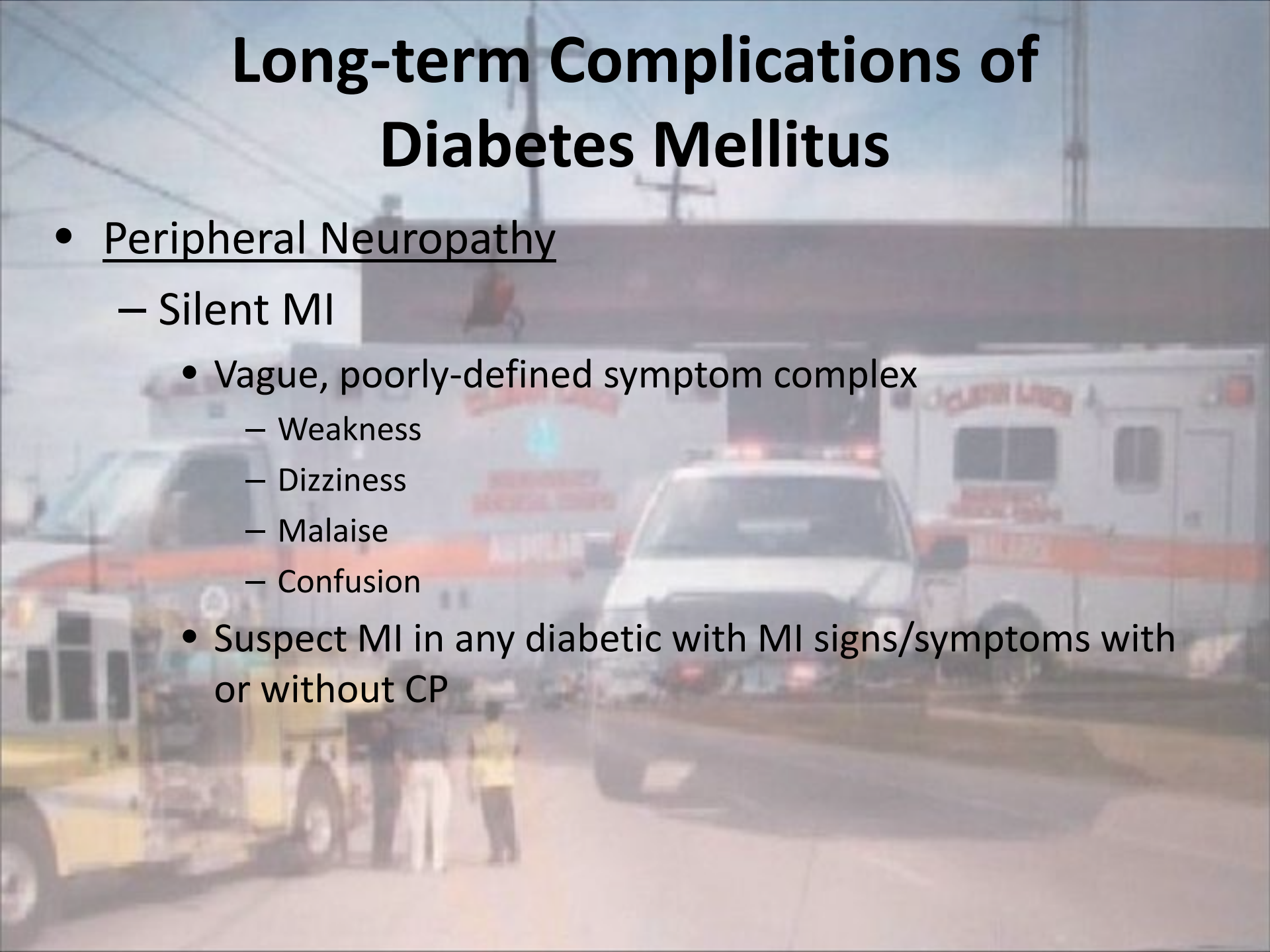
- Peripheral Neuropathy

- Silent MI

- Vague, poorly-defined symptom complex

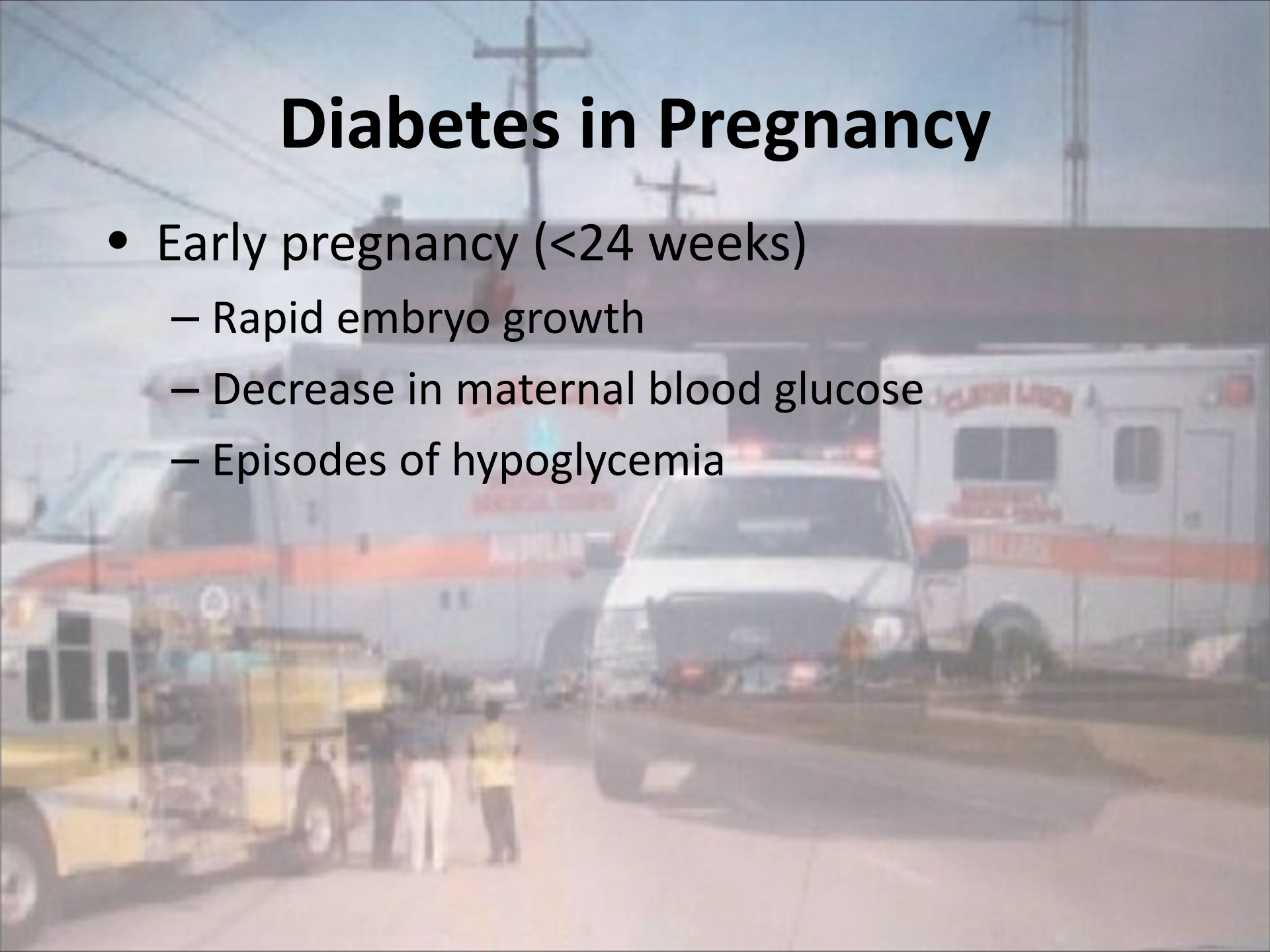
- Weakness
- Dizziness
- Malaise
- Confusion

- Suspect MI in any diabetic with MI signs/symptoms with or without CP



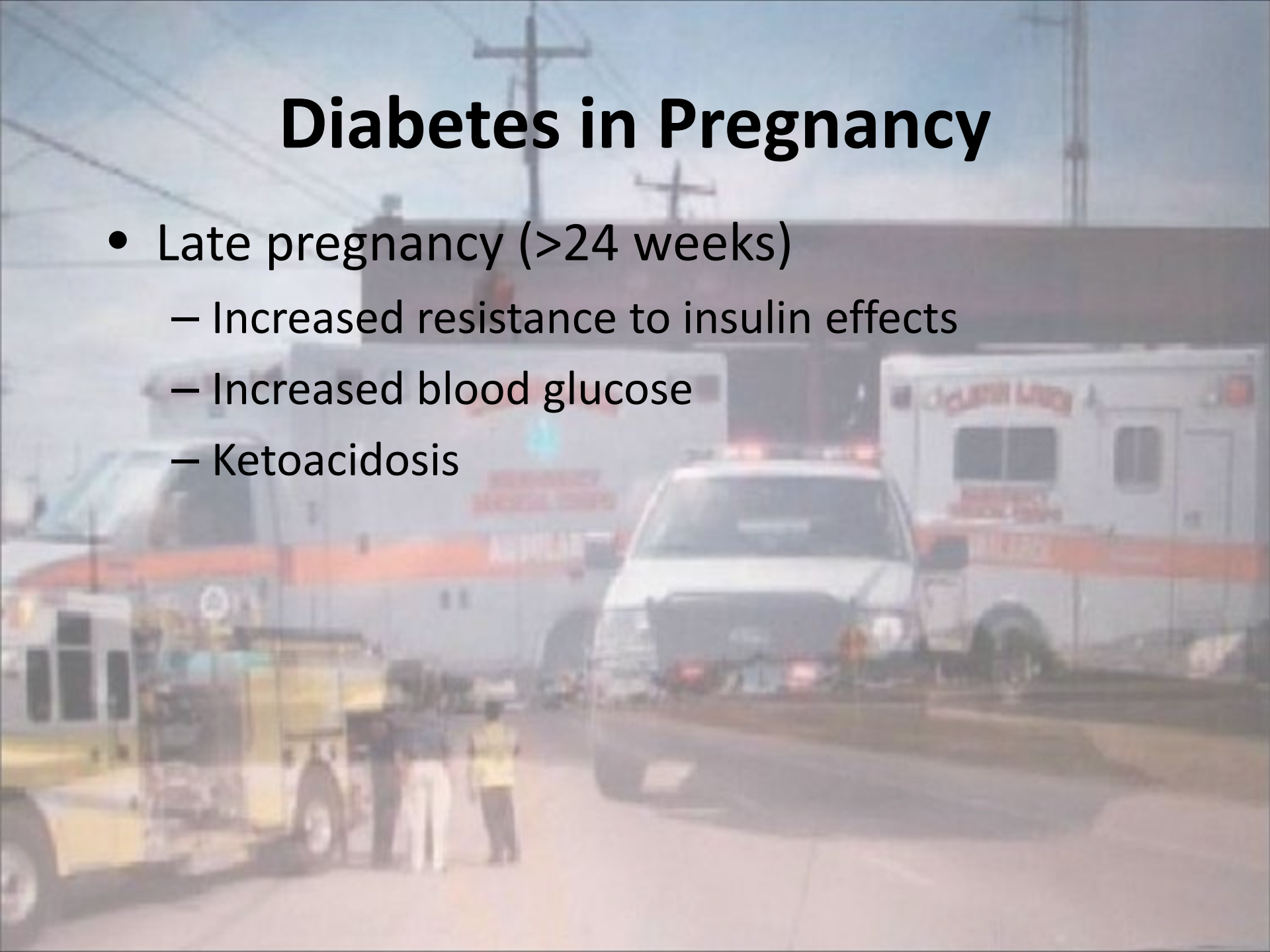
Diabetes in Pregnancy

- Early pregnancy (<24 weeks)
 - Rapid embryo growth
 - Decrease in maternal blood glucose
 - Episodes of hypoglycemia



Diabetes in Pregnancy

- Late pregnancy (>24 weeks)
 - Increased resistance to insulin effects
 - Increased blood glucose
 - Ketoacidosis



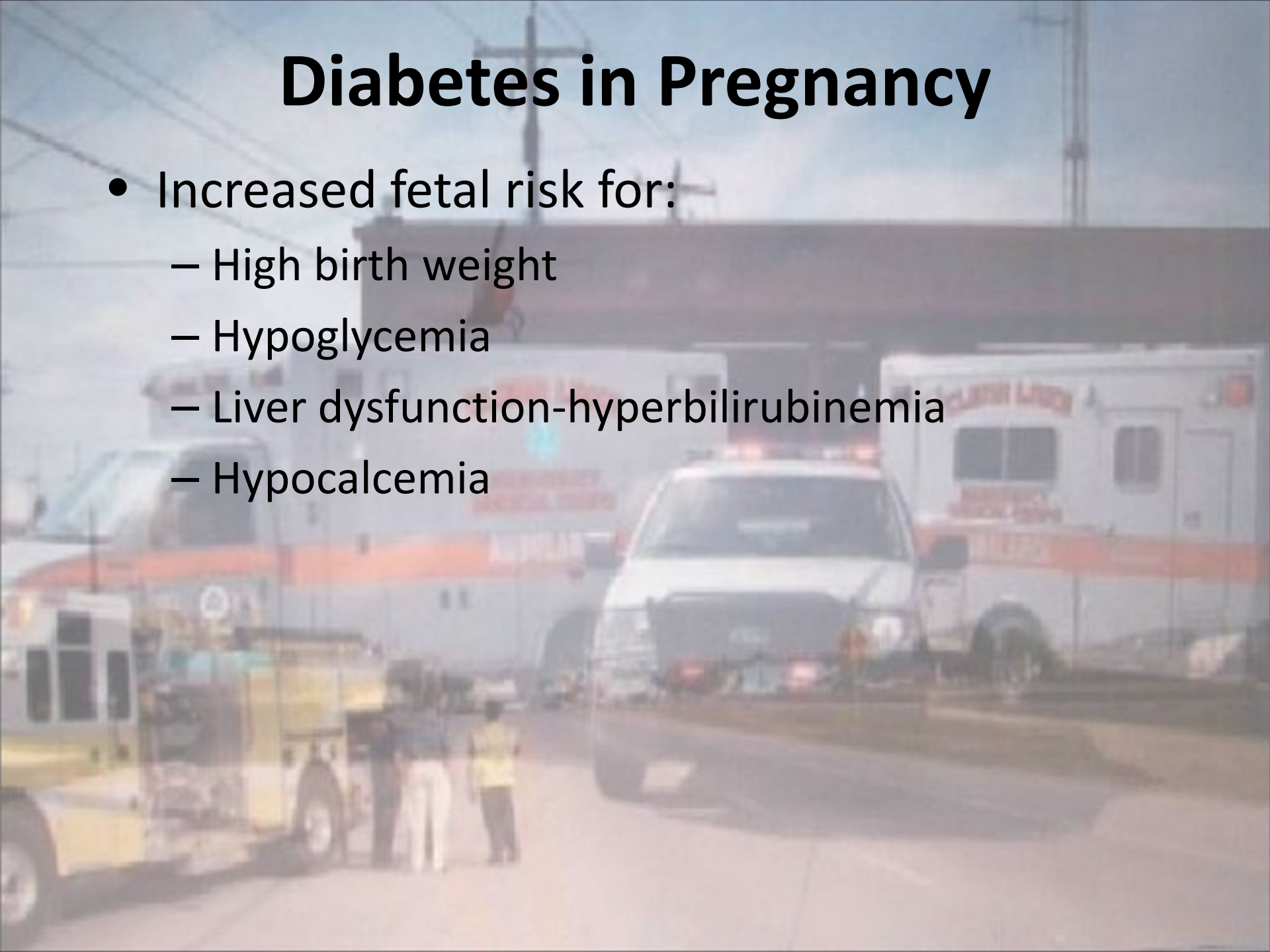
Diabetes in Pregnancy

- Increased maternal risk for:
 - Pregnancy-induced hypertension
 - Infections
 - Vaginal
 - Urinary tract



Diabetes in Pregnancy

- Increased fetal risk for:
 - High birth weight
 - Hypoglycemia
 - Liver dysfunction-hyperbilirubinemia
 - Hypocalcemia



Assessment of the Diabetic Patient

- Maintain high-degree of suspicion
- Assess blood glucose level in all patients with
 - seizure, neurologic S/S, altered mental status
 - vague history or chief complaint
- Blood glucose assessment IS NOT necessary in all patients with diabetes mellitus!!

Assessment of the Diabetic Patient

- History and Physical Exam includes
 - Look for insulin syringes, medical alert tag, glucometer, or insulin (usually kept in refrigerator)
 - Last meal and last insulin dose
 - Missed med or missed meal?
 - Signs of infection
 - Foot cellulitis / ulcers
 - Recent illness or physiologic stressors

Blood Glucose Assessment

- Capillary vs. venous blood sample
 - Depends on glucometer model
 - Usually capillary preferred
- Dextrostick vs Glucometer
 - Dextrostick - colorimetric assessment of blood provides glucose estimate
 - Glucometer - quantitative glucose measurement
- Neonatal blood
 - Many glucometers are not accurate for neonates

This Concludes Diabetes Mellitus

Please visit the link below to take the EXAM

If you have any questions please contact:

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or

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