Chapter one

1.1 Introduction

The pancreas is one of the juices and enzymes production organ located deep in the abdomen and situated behind the stomach. The enzymes produced by the pancreas help in digesting fat, protein, and carbohydrates before being absorbed by the intestine. The pancreas also produces insulin, which is important in regulating the glucose concentration in the blood. Any system dysfunction or irregularity occurs to the pancreas may lead to several diseases such as diabetes mellitus, acute pancreatitis, chronic pancreatitis, pancreatic enzyme deficiency as well as pancreas tumor.(Daniel S, etal, 2014).

There are two types of DM: type Iwhich is an autoimmune disorder with infiltrationof inflammatory cells in Islets of Langerhansand distraction of pancreatic beta cells 3and type II which is characterized with disturbancein insulin secretion, peripheral resistanceto insulin and overproduction of glucose byliver. Pancreas as the insulin-producing glandis changed and destroyed in the process thatleads to diabetes. Pancreatic markers in type Idiabetic patients are infiltration of inflammatorycells in islets showing chronic inflammationand production of new beta cells.The ultrasound is a non- invasive imaging tool that send the sound and receive the echoes to visualize in great detailorgans. The location of the pancreas in the abdomen makes it well suited for ultrasound examination. However, the complex anatomy of the organ and surrounding tissues make evaluation a demanding task, and the ultrasound echo of even the normal pancreas varies widely from patient to patient.(Daniel S. et al 2014).

Ultrasonography (U/S) of pancreas is a common imaging study performed for diagnosis the pancreatic disease; it’ssafe, convenient ,and relative inexpensive.

There are many studies of measured the pancreas in diabetic patient ,M Gouldet, al2005 and Alzaid A et,al 2000 therestudied size of pancreas in diabetic patient based ultrasound., they were measured the head and body of pancreas and the effect the different types and the duration of diabetic on pancreatic size, and in this study is going to measure the head, body and the tail of pancreas.And assess the effect of duration of diabetic on the pancreassize in theSudanese patient. (Daniel S. et al 2014).

1.4 Problem of the study:

The Diabetic has become very widespread in the Sudan between all age groups may lead to a wide difference from the normal condition, and the pancreas specially is have greats changes so that the size of pancreas so difficulty to measurements by laboratories investigations.

1.5 Objectives:

1.5.1 General objective:

To assess the pancreatic size in diabetic patient using ultrasonography

1.5.2Specific objectives:

- To measure the size ofpancreatic(the head, body and tail) in diabetic patient
- To determine the association between the size of the pancreas and the diabetic.
- To estimate the relationship between the duration of diabetes and pancreas size.
- To test the difference in pancreatic size between the normal and the diabetic patient.

- To compare the findings of this study with another studies.

1.6 Significant of the study:

To give knowledge and full information about the effect of diabetes on the pancreas morphology, and the percentage change of pancreatic size related to the diabetic and duration of diabetic and the age of the patient.

-the finding of this study can help the physians, specialist to diagnosis and following the diabetic.

1.7 Overview of study:

This study was consists of five chapters; Chapter one included the introduction of this thesis, problem of the study, objectives (general and specific), significant of the study and the overview of the study. Chapter two concerned with literature review, also was include general theoretical background of this topic.Chapter three dialed with themethods and materials that used on this study, chapter four showed the result ofthe study. Chapter five was including the discussion, conclusion and recommendation in addition to the references and appendices.

Chapter Two

Literature review

2.1 Anatomy of pancreas:

The pancreas lies in the upper abdomen behind the stomach. The pancreas is part of the gastrointestinal system that makes and secretes digestive enzymes into the intestine, and also an endocrine organ that makes and secretes hormones into the blood to control energy metabolism and storage throughout the body.It is worthwhile to mention a few definitions for key terms as used in the context of the pancreas:Exocrine pancreas, the portion of the pancreas that makes and secretes digestive enzymes into the duodenum. This includes acinar and duct cells with associated connective tissue, vessels, and nerves. The exocrine components comprise more than 95% of the pancreatic mass. And endocrine pancreas, the portions of the pancreas (the islets) that make and secrete insulin, glucagon, somatostatin and pancreatic polypeptide into the blood. Islets comprise 1-2% of pancreatic mass.Since we are dealing with a three dimensional solid structure, the aphorism that “a picture is worth a thousand words” seems to pertain accordingly, this chapter will largely consist of images with extended legends. The images range from classic work of skilled medical artists to original drawings and photomicrographs from leaders in the study of pancreatic anatomy and structure*(Daniel S, et al, 2014).*

2.1.1 Gross Anatomy:

Figures 2-1 depict the gross anatomy of the pancreas and its relationship to surrounding organs in adults. It is customary to refer to various portions of the

pancreas as head, body, and tail. The head lies near the duodenum and the tail extends to the hilum of the spleen.When the terms anterior, posterior, front and back are used, they pertain to relationships in the human, standing erect. Superior and inferior are used in the same context so that they mean toward the head and toward the feet, respectively. These usages obviously do not pertain in quadraped animals where dorsal, ventral, cephalad, and caudal are more useful terms.(Daniel S, etal, 2014). Use of the terms left and right can be problematic. For example, the spleen is located in the upper portion of the abdomen on the left side of the body. When the abdomen is pictured from the front, this places the spleen on the viewer's right hand side. We will adopt the convention that right and left (unqualified) will be used in the first sense in the legends for gross anatomy (indicating the subject's right and left side). When we are designating location within an image, we will use "image right" and "image left" to denote relationships within the image*(Daniel S, et al, 2014).*

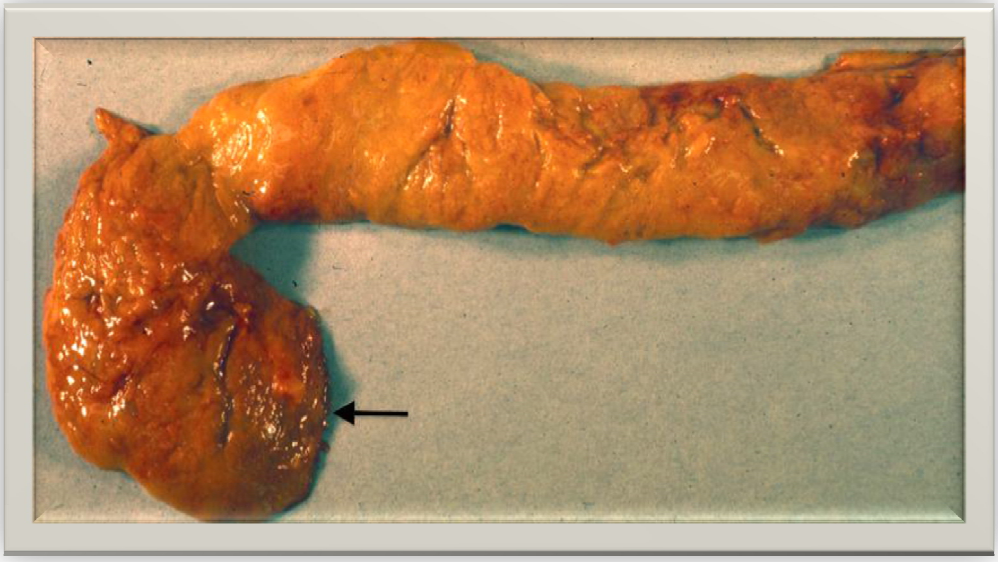


Figure.2.1the gross anatomy of the human pancreas (Daniel S, etal, 2014).

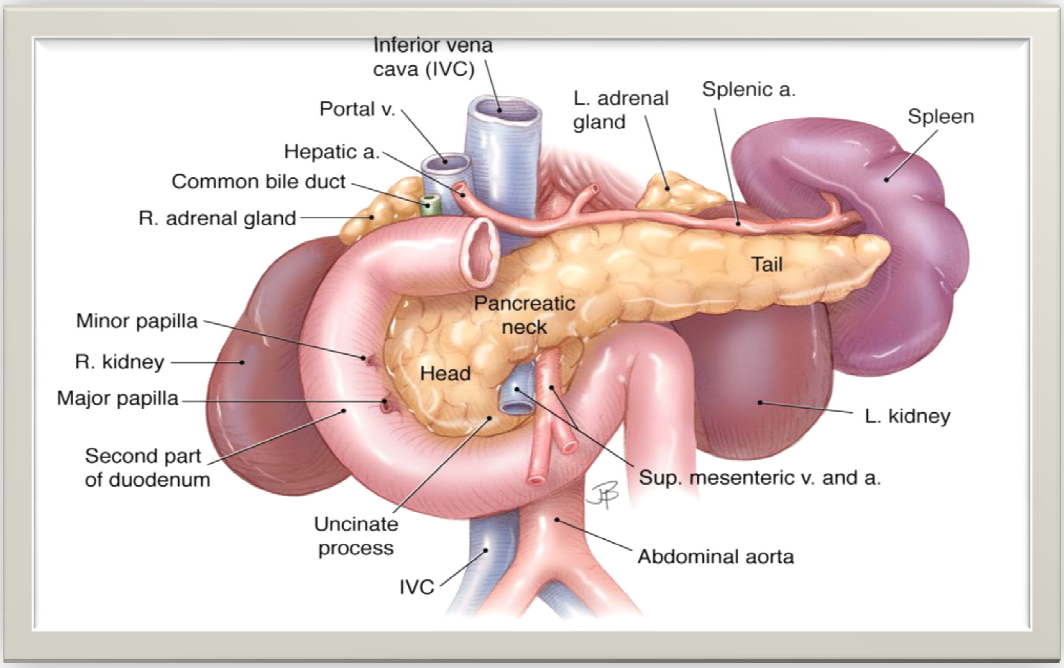
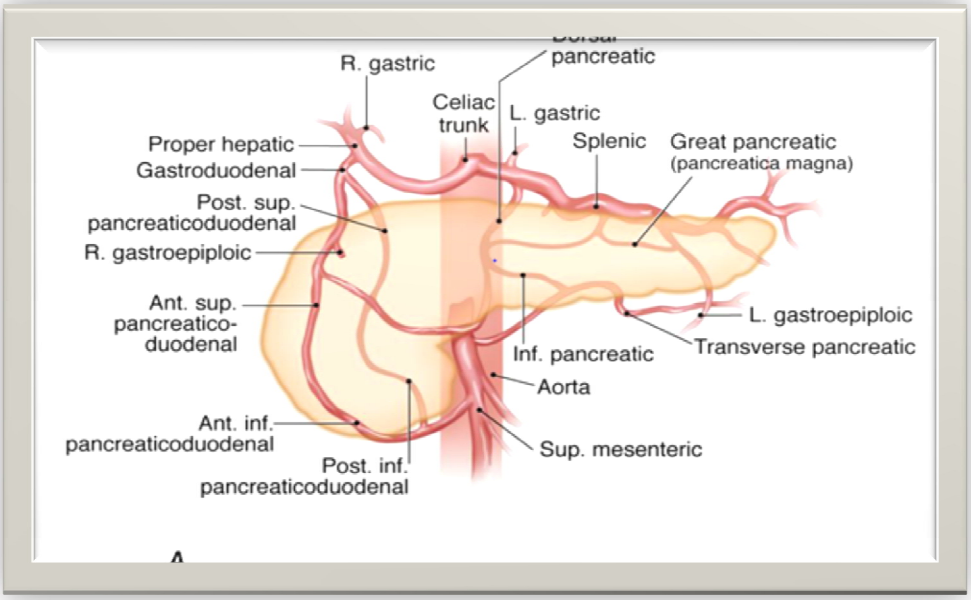


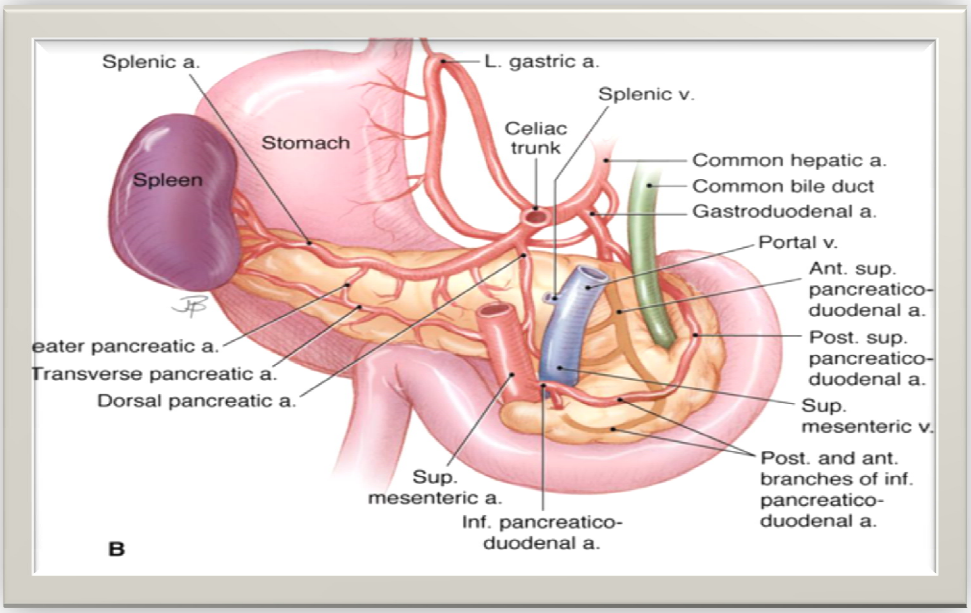
Figure.2.2 Anatomic relationships of the pancreas with surrounding organs and structures (Daniel S, et al, 2014).

Several key relationships should be noted. Their recognition may be facilitated by also referring to Figure(2.2). The head of the pancreas lies in the loop of the duodenum as it exits the stomach, The tail of the pancreas lies near the hilum of the spleen, The body of the pancreas lies posterior to the distal portion of the stomach between the tail and the neck and is unlabeled in this drawing, The portion of the pancreas that lies anterior to the aorta is somewhat thinner than the adjacent portions of the head and body of the pancreas. This region is sometimes designated as the neck of the pancreas and marks the junction of the head and body, The close proximity of the neck of the pancreas to major blood vessels posteriorly including the superior mesenteric artery, superior mesenteric-portal vein, inferior vena cava,

and aorta limits the option for a wide surgical margin when pancreatectomy(surgical removal of the pancreas) is done, The common bile duct passes through the head of the pancreas to join the main duct of the pancreas near the duodenum as shown in Figure(2-2). The portion nearest the liver lies in a groove on the dorsal aspect of the head (see Figure2.2).and the minor papilla where the accessory pancreatic duct drains into the duodenum and the major papilla (ampulla of Vater) where the main pancreatic duct enters the duodenum are depicted.(DonielS et,al 2014).



(A)



(B)

Figure 2.3.The arterial blood supply of the pancreas. (Doniel S, etal, 2014).

The upper panel (A) is visualized from the front, and the lower panel (B) is seen from the back. The celiac trunk and the superior mesenteric artery both arise from the abdominal aorta. Both have multiple branches that supply several organs including the pancreas. The anastomosis of their branches around the pancreas provides collateral circulation that generally assures a secure arterial supply to the pancreas. Most of the arteries are accompanied by veins (not shown) that drain into the portal and splenic veins as they pass behind the pancreas as shown in B. The superior mesenteric vein becomes the portal vein when it joins the splenic vein.

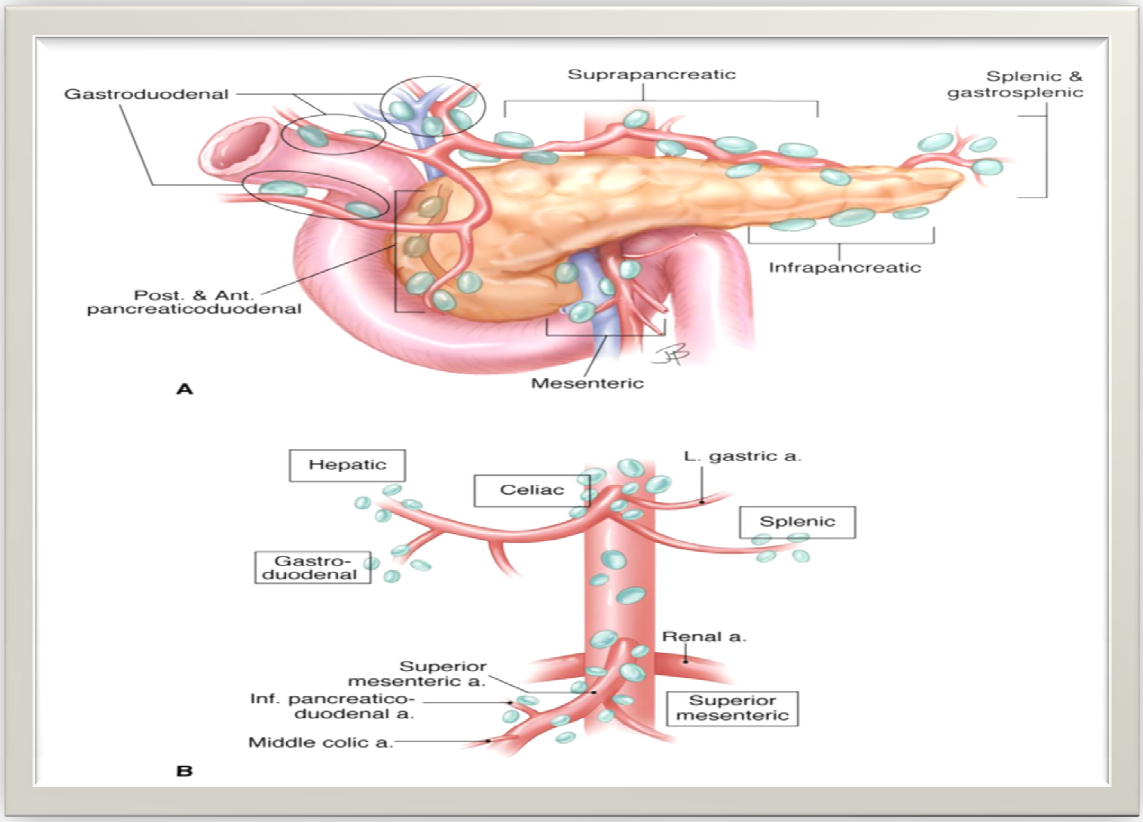


Figure2.4. Lymph nodes draining the pancreas(Daniel S, etal 2014)

This figure indicates the typical location of lymph nodes surrounding the pancreas. There is considerable individual variation in the location of lymph nodes and an

image like this is idealized. Both A and B are anterior views. B includes some nodes that lie posterior to the pancreas. Image by Jennifer Parsons Brumbaugh used with permission of the publisher [Daniel S etal 2014].

2.1.2 Embryology and Development

The pancreatobiliary anlagen appear at gestation week 5 in the human; fusion of the dorsal and ventral anlagen occurs during week 7(Gilbert et al 2004).

Full development of acinar tissue extends into the postnatal period. In mice, pancreatic development begins at embryonic day 8.5 and is largely complete by day e14.5 (Gilbert, et al, 2004).

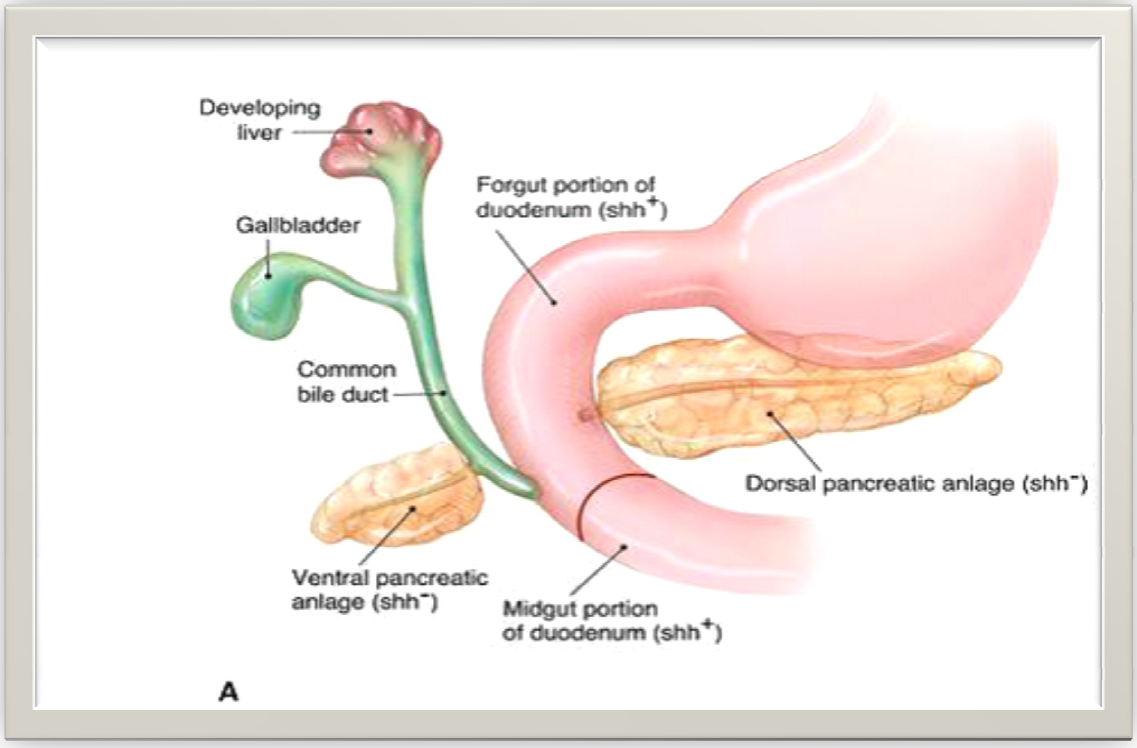


Figure2.5. the figure reflects the embryonic development of the pancreas and biliary system in the human.(Gilbert, et al, 2004).

The pancreas develops from two outgrowths of the foregut distal to the stomach. The ventral diverticulum gives rise to the common bile duct, gallbladder, liver and the ventral pancreatic anlage that becomes a portion of the head of the pancreas with its duct system including the uncinate portion of the pancreas. The dorsal pancreatic anlage gives rise to a portion of the head, the body, and tail of the pancreas including a major duct that is continuous through the three regions.The caudal portion of the head of the pancreas (uncinate) and the major papilla (ampulla of Vater) are derived from the ventral anlage. The minor papilla that drains the duct of Santorini is derived from the dorsal anlage. Image by Jennifer Parsons Brumbaugh used with permission of the publisher (Gilbert, et al, 2004).

2-2 physiology of pancreas:

The pancreas is an integral part of the digestive system, and the flow of the digestive system is often altered during the surgical treatment of pancreatic cancer. Therefore it is helpful to review the normal flow of food and the flow of digestive enzymes of the pancreas before reading about surgical treatment.Food is carried from the mouth to the stomach by the esophagus. The esophagus is a tube that descends from the mouth down into the abdomen. In the abdomen, the esophagus empties into the stomach where digestive acids made by the stomach break down the food. From the stomach, the partially digested food flows directly into the first part of the small intestine, called the duodenum. It is here in the duodenum that bile from the liver and digestive enzymes from the pancreas enter the digestive system.*(Foulis AK, etal,2005*).

The duodenum then leads to the other parts of the small bowel, the jejunum and ileum, where further digestion of food takes place. The ileum then empties into the

large bowel (also known as the large intestine), and finally completely digestive material passes out of the body through the anus.*(Foulis AK, etal,2005).*

The Exocrine Function:The pancreas contains exocrine glands that produce enzymes important to digestion. When food enters the stomach, these pancreatic juices are released into a system of ducts that culminate in the main pancreatic duct. The pancreatic duct joins the common bile duct to form the ampulla of Vater which is located at the first portion of the small intestine, called the duodenum. The common bile duct originates in the liver and the gallbladder and produces another important digestive juice called bile. The pancreatic juices and bile that are released into the duodenum, help the body to digest fats, carbohydrates, and proteins].Endocrine Function:The endocrine component of the pancreas consists of islet cells that create and release important hormones directly into the bloodstream. Two of the main pancreatic hormones are insulin, which acts to lower blood sugar, and glucagon, which acts to raise blood sugar. Maintaining proper blood sugar levels is crucial to the functioning of key organs including the brain, liver, and kidneys*(Foulis AK, etal,2005)*

2-2-1Physiology of insulin secretion:

The B cell of the pancreas secretes equimolar amounts of C-peptide and insulin. In the normally functioning B cell, the vast majority (> 95 %) of proinsulin is converted toinsulin and C-peptide before release and the proinsulin to insulin ratio is less than 5 %.Approximately, 50 to 60 % of the insulin secreted by the pancreas intothe portal system is removed during its initial passage through the liver(first pass effect) thatleads to a 2-3 timesgreater concentration of insulin in the portalvein compared with a peripheral vein. Basalinsulin secretion is critical to the maintenance of basal euglycaemia. Quantitatively, pancreatic insulin secretion

inthe basal state varies from 0.25 to 1.5 U/h in normal subjectsand accounts for 50 % or more of the 24-hour integrated insulin secretion. Evidence exists forrapid oscillations in the basalinsulin levels with periods of9 to 14 minutes .*(Alzaid A etal 2006).*

Severalstudies have convincingly shown that equal amountsof insulin presented to target organs haveimproved action when delivered in a pulsatile manner .Both basal insulin secretion and meal-related insulin secretion are regulated byglucose, although other nutrients (amino acids,fat-derived products), hormones (glucagon,gastric inhibitory polypeptide,glucagon-like peptide-1 or GLP-1) and neural factors affectinsulin release. The intravenous glucose tolerance test (IVGTT) is classically used forevaluating glucose-regulated insulin secretion and B-cell function. In response to intravenousglucose, insulin is releasein a biphasic pattern The first phase (acute) insulinresponse to glucose (AIRglucose) begins within 1 minute after an IV glucose bolus, peaksbetween 3 and 5 minutes, and lasts up to 10 minutes..*(Alzaid A et al 2006*).

The insulin released from the pancreasduring this first phase has already been synthesized and stored inthe secretory granules of theB cell. The second phase insulin response to glucose begins just after the glucose bolus (but isnot evident until 10 minutes later) and lasts as long as the hyperglycaemia persists (so it ismuch more sustained during anhyperglycaemic clamp than after an IVGTT). This secondphase insulin response depends primarily on insulin stores, but is also regulated by newprotein synthesis within the Bcell. Unlike the first phase, thesecond phase insulin release isdirectly proportionate to the steady state glucose concentration immediately preceding theglucose bolus. Thus, when evaluating second phaseinsulin responses it iscritical to achievecomparable steady glucose concentrations as best obtained during a hyperglycaemic glucose clamp. While the oral glucose tolerance test (OGTT) is a good measure of overall glucose tolerance,

it is less accurate to evaluated insulin secretion ad B-cell function. Nevertheless, the so-called insulin genic index (delta insulin0-30 min/delta glucose 0-30 min) has been proposed to evaluate early insulin response and has been shown to be highly related to the AIR glucose during an IVGTT. A strong negative relationship has been demonstrated between cephalic insulin release and initial glucose increment after a glucose load, suggesting that an early surge of insulin secretion could exert a restraining effect on rising blood glucose. .*(Alzaid A et al 2006).*

Furthermore, an inverse correlation was reported between plasma insulin levels 30 min after an oral glucose load and the plasma glucose concentration attained in the second hour, suggesting that the effects of an acute (first-phase) insulin secretion significantlyimpact subsequent glucosetolerance. The amount of insulin released during first-phasesecretion suggests a more likely effect on the liver (inhibition of glucose hepatic output)than on peripheral tissues (stimulation of muscular glucose uptake). The large insulin response to meal intake, in spite of the moderate increase in circulating glucose levels, is dueto the so-called incretin action. This effect is due to gut hormones ("incretins") that are released intothe circulation following meal ingestion and stimulate insulin secretion post-prandially. One of the most importantincretins is GLP-1.*(Alzaid A et al 2006).*

Interestingly, several animal studies demonstrated that GLP-1 may increase insulin secretion not only by promoting B-cellfunction through a direct insulino-tropic action, but also through stimulation of B-cell mass by augmenting B-cell proliferation and inhibiting B-cell apoptosis. This might be important for the concept of treating type 2 diabetes. It has been demonstrated that the magnitude of AIR to non-glucose stimuli (such as arginine, glucagon or epinephrine) is dependent of the prestimulus glucose level. The slope of the straight line relating the magnitude of AIR to the ambient plasma glucose level at which it was measured is defined as the slopeof glycaemic potentiation. Although the slope of

potentiation can be a useful measure of B-cell function, it is a combined measure that isinfluenced both by changes in insulin secretary capacity and changes in sensitivity of the B cell to the potentiating effects of glucose. .*(Alzaid A et al 2006).* To determine which of these two factors is responsible for the changes in slope, one must complete the dose-response curve and from it calculate two parameters : AIRmax or maximal acute insulin response is a measure of the insulin secretory capacity of the pancreas whereas PG-50, defined as the ambient glucose level at which the half-maximal AIR response occurs, is an index of the sensitivity of the B cells to the potentiating effects of glucose (PG-50 and glucose sensitivity are inversely related) .It is noteworthy that islet B-cell function is regulated by tissue insulin sensitivity.Evaluation of the relationship of islet function to insulin sensitivity suggests that in glucose-tolerant subjects a reciprocal relationship exists in which insulin secretory capacity and glucose potentiation of insulin secretion increase to compensatefor decreasing sensitivity of the peripheral tissues to insulin Thisability to adapt may explain why states of insulin resistance such as obesity are characterized byhyperinsulinaemia with little hyperglycaemia. .*(Alzaid A et al 2006).*

This finding suggests that insulin resistance is a regulator of insulin secretion and that a component of this regulatory mechanism involves enhanced B-cell responsiveness to glucose. Intact cross-talk betweeninsulin secretion and insulin action persists after post-gastroplastyrecovery of ideal body weight in severely obese patients. Interestingly, in contrast to the findings in obesity, the B cell does not appear to adapt to the insulin resistance of aging. The frequency ofglucose intolerance inolder individuals may therefore be determined in part by this relativeimpairment of islet function. The hyperbolic relationship between insulin sensitivity and insulin responses has important implications for estimating the adequacy of a B-cell response in humans. As differences in insulin sensitivity must

be balanced by reciprocal changes ininsulin release in order to maintain glucose tolerance, it is apparent that although insulin responses may be identical in groups of subjects, if insulin sensitivity is not the same, B-cell function is different. Thus, it is essential to consider insulin sensitivity and insulin responses together when evaluating B-cell function and when assessing the importance of B-cell function to glucose tolerance after both intravenous and oral stimulation. .*(Alzaid A et al 2006).*

2-3Pathology of pancreas:

2-3-1Pathophysiology of insulin secretion in type 1 diabetes :

The majority of patients who have had type 1 diabetes for more than 3 have essentially no endogenous insulin secretion either in the basal state or in response to meals. While the clinical onset of type 1 diabetes is usually abrupt and dramatic, it is now known that the underlying autoimmune destructive process may take place over months or years before the overt clinical presentation.

(Altobelli E etal 2008).

During this time the individuals are asymptomatic and have first normal glucose tolerance that progressively deteriorates to impaired glucose tolerance. The earliest marker of impaired B-cell function in type 1 diabetes appears to be a loss of the ability of glucose to potentiate non-glucose secretagoguesThe next detectable abnormality is loss of the acute (or first phase) insulin response tointravenous glucose. By the time that clinical type 1 diabetes is present, the usual pattern of B-cell function is for individuals to have no potentiating slope, lack of first and second phase insulin response to glucose, low to normal fasting insulin level, and a reduced but still detectable insulin response to intravenous glucagon or arginine. Several studies confirm that although some endogenous insulin secretion is usually present at the onset of clinical diabetes (positive C-peptide), its subsequent rate of decline may vary considerably. *(Altobelli E et al 2008).*

The rate of decline is classically much faster in children and adolescents than it is in adults. In general, stimulated C-peptide is about 20 % of normal at the onset of clinical type 1 diabetes, in agreement with animal studies suggesting that over 80 % of the functioning B cells need to be destroyed before overt diabetes will develop.Whether B-cell death in type 1 diabetes is due to apoptosis or necrosis or a combination of both has not been clarified. The almost inevitable subsequent loss of B cells may be slowed to some extent by attention to strict metabolic control. Following initiation of insulin therapy, numerous patients undergo a partial remission, during which insulin doses may be markedly reduced or even insulin therapy can be withdrawn completely. This so called “honeymoon period” occurs largely because of a transient improvement in B-cell function, along with an improvement in insulin sensitivity. *(Altobelli E et al 2008).*

The mechanism responsible for this improvement in insulin secretion is unknown. One theory is that chronic severe hyperglycemia overdrives the residual B cells and desensitisesor exhausts them. Then, treatment with exogenous insulin lowers the plasma glucose level, reducing the drive and allowing the residual B cellsto recover and to regain partial function. Unfortunately the honeymoon period is usually transient (this phase rarely lasts more than a few months), perhaps because of continuing autoimmune damage of the residual B-cell population. Even after treatment with exogenous insulin is once again instituted, the presence of some endogenous insulin secretion has been shown to reduce glycemic swings and improve the overall metabolic control. Recent data from the Diabetes Control and Complications Trial (DCCT) demonstrated that even modest levels of B-cell activity at entry in the study were associated with reduced incidences of retinopathy and nephropathy, while continuing C-peptide (insulin) secretion is also

important in avoiding severe hypoglycemia, the majorcomplication of intensive insulin therapy. *(Altobelli E et al 2008).*

Attempts have therefore been made to preserve endogenous insulin secretion in newly diagnosed type1 diabetic patients using various immunosuppressive or other techniques, but so far success has been limited. If apoptosis is the main common mode by which B cells die in response to immune attack by cytokines and/or T-cells in type 1 diabetes, it may be possible to develop novel strategies to prevent this mode of B-cell death, thereby preventing or delaying the onset of the disease .New therapeutic strategies can restore B-cell function in type 1 diabetic patients and those patients can be free from insulin therapy. B-cell replacement therapy could be by whole-pancreas transplants, islet transplants, implants of insulin-producing cells, or induction of ectopic expression of insulin. Whereas the first is a reality and the second has had recent promising success, the other strategies are being explored in experimental animals. *(Altobelli E et al 2008).*

However, it appears that insulin secretion capacity may progressively decrease with time, whatever the method used for B-cell replacement. Therefore, further efforts should be made to better know the pathophysiology of these new sources of insulin release in order to preserve insulin secretion and protect against metabolic deterioration in such individuals *(Altobelli E etal 2008).*

2-3-2Pathophysiology of insulin secretion in type 2 diabetes:

Type 2 diabetes accounts for 80-90 % of all diabetes in most countries. However, diabetes is a far more heterogeneous disease than the current classification into type 1diabetes and type 2diabetes implies (Fonseca V et al 2010).

About 10 % of Caucasian patients with presumably type 2 diabetes have glutamate acid decarboxylase (GAD) antibodies or so-called latent auto-immune

diabetes in adults (LADA). In addition, among patients with early onset of familial diabetes younger than 40 years, about 15 % carry mutations in MODY (Maturity Onset Diabetes of the Youth) or mitochondrial genes. It has been demonstrated that carriers ofMODY mutations (even glucose-tolerant individuals) are characterized by a severe impairment of insulin secretion .In patients with newly diagnosed type 2 diabetes inthe UKPDS, autoantibody detection (identifying patients with LADA) has been shown more powerful to predict subsequent insulin dependency than other classical predictive factors using stepwise logistic regression analysis, and this observation has been confirmed in other Scandinavian studies.(*Fonseca V et al 2010).*

These data clearly emphasize the heterogeneous nature of "type 2" diabetes and stress the importance of defining the underlying subtypes when studying the pathogenesis of the disease, and especially the respective contributions of defects in insulin secretion and insulin sensitivity Controversy exists regarding the primacy of the contribution of defects in insulin sensitivity versus insulin secretion to the development of type 2 diabetes. Basal insulin concentrations in patients with type 2 diabetes have been reported as either elevated, reduced or normal. In addition, cross-sectional studies examining plasma insulin responses during have found an inverted U relationship between the 2-h plasma glucose level (a generally recognized index of glucose tolerance) and the 2-h plasma insulin level. This pattern has been interpreted to indicate that early on, as glucose tolerance decreases, there is increased insulin secretion and, therefore, that insulin resistance, rather than insulin deficiency, is responsible for the development of impaired glucose tolerance. (*Fonseca V et al 2010).*

However, it is difficult to compare fasting insulin levels of different individuals unless they are matched for both the steady state glucose concentration and body

adiposity (as a marker of insulin resistance) .When such matching is performed, it is evident that there is a marked insulin secretory defect in all patients with type 2 diabetes. Thus, hyperinsulinaemia, even if appropriate for the prevailing hyperglycaemia, does not necessarily indicate normal B-cell function. In addition, the interpretation of OGTT data may be questioned as it does not take into consideration the importance of the kinetics of insulin release and the dependence of insulin secretion upon the prevailing plasma glucose concentrations. Clearly there is a progressive decrease in the early (30 min) plasma insulin response as glucose tolerance deteriorates.*(Fonseca V et al 2010).*

These and otherobservationsprovide evidence thatthere is impaired pancreatic B-cell function before the onset of impaired glucose tolerance (IGT) and that late hyperinsulinaemia may actually be the result of an inadequate B-cell response to the hyperglycaemia due to impaired early insulin release and may not necessarily indicate the presence of insulin resistance. Thus, the misleading dichotomy established between insulin deficiency (versus impaired insulin release) and insulin resistance has led to a general underemphasis of the issue of the appropriateness of B-cell function. It has become clear that B-cell function should be interpreted in the context of the degree of insulin sensitivity.*(Fonseca V et al 2010).*

Failure to take into account the hyperbolic relationship between B-cell function and insulin sensitivity has been misleading. By accounting for this interaction, it has been clearly demonstrated that subjects at high risk for developing type 2 diabetes (older individuals, women with a history of gestational diabetes or polycystic ovary syndrome, subjects with impaired glucose tolerance) have impaired B-cell function. Despite the fact that it has long]been the prevalent view that insulin resistance is the main genetic factor predisposing to development of type 2 diabetes, the recent literature better supports the case of impaired insulin secretion being the initial and main geneticfactor predisposing to type 2 diabetes.

Evidence to support this view can be found especially in studies in people at high risk to subsequently develop type 2 diabetes as discordant monozygotic twins or first-degree relatives of patients with type 2 diabetes. *(Fonseca V et al 2010).*

In patients with IGT or in the early stages of type 2 diabetes, first phase insulin releaseis almost invariably lost. This defect results in an impaired inhibition of endogenous (liver) glucose production and plays a pathogenic role in post-mealhyperglycaemia. Therefore, the restoration of the dynamics of insulin secretion following a meal should be seen as a rational therapeutic approach in the treatment of type 2 diabetes because it induces an overall improvement in glucose tolerance with the advantage of possibly lowering chronic hyperinsulinaemia. Whereas type 2 diabetes is characterized by an absent first phase insulin release to intravenous glucose, second phase insulin release is still sensitive to glucose and therefore partially maintained in patients with compensated diabetes. These responses are maintained by hyperglycaemia until plasma glucose canno longer rise sufficiently to compensated for impaired insulin secretion. Generally decompensation occurs in patients with fasting plasma glucose levels greater than 200-250 mg/dl].*(Fonseca V et al 2010).*

The United Kingdom Prospective Diabetes Study (UKPDS) clearly showed that type 2 diabetes is a progressive disorder, and that the relentless decline in glycaemic control over years is undoubtly related to a decrease in B-cell function. We previously reported that insulin secretion decompensation (as assessed during an intravenous glucagon test) in face of insulin resistance (as assessed during a euglycaemichyerinsulinaemic clamp) and further progressive failure explain much of the natural type 2 diabetes and the progression towards insulin requirement.Indices of B-cell function that are independent of plasma glucose level or that account for the effect of hyperglycaemia reveal a marked impairment of insulin secretion early in type 2 diabetes. Alterations in pulsatile insulin release and

ultradian oscillatory insulin secretion can be observed .The B cell is also unable to oscillate in concert with the fluctuations in plasma glucose induced by an oscillating glucose infusion. Inefficient proinsulin processing to insulin leads to increased proinsulin to insulin ratio. *(Fonseca V et al 2010).*

This abnormality does not appear to be secondary to the increased secretory demand but rather reflects a yet-undefined abnormality of B-cell function in the insulin secretory process. A reduction in the release of islet amyloid polypeptide (IAPP-, known also as amylin) has been observed in established type 2 diabetes. Finally, the slope of glycaemic potentiation and AIR max are both markedly reduced whereas PG-50 is normal. Thus, there is a major reduction in the capacity of type 2 diabetic patients to secrete insulin despite normal sensitivity of the B cells to the potentiating effects of glucose .The mechanism for the defect in glucose regulation of insulin secretion in subjects with type 2 diabetes is largely unknown .Some data suggest that the islet mass is reduced and therefore the capacity of the pancreas to secrete insulin is diminished in type 2 diabetes. Recent observations showed that the major defect leading to a decrease in B-cell mass in type 2 diabetes is increased apoptosis. However, the reduction is not sufficient to explain the marked defect in insulin secretion. *(Fonseca V et al 2010).*

The functional B-cell loss exceeds the expected impact of a 20-50 % loss of B cells reported at autopsy. This loss may be explained by the simultaneous deposition of amyloid, a product of human IAPP normally produced in the B cell and secreted along with insulin. It has been hypothesized that the process of amyloid fibril formation impairs function early (as evidenced by disproportionate hyperproinsulinaemia) and leads to late B-cell failure and eventual death [Fonseca V et al 1985]. Studies to impair such fibril formation offer the possibility of developing preventive means for the relentless downhill course of the disease, which is one of the most important current clinical problems in type 2 diabetes

management. Recent animal findings with B-cell-specific disruption of the insulin receptor suggest that impaired insulin secretion might be a result of insulin resistance in the B cells themselves.*(Fonseca V et al 2010).*

The loss of first-phase insulin secretion after glucose challenge seen in these BIRKO (Beta cell Insulin Receptor Knock Out) mice resembles what is seen in human type 2 diabetes. These data suggest that signals mediated by the insulin receptor are essential for the release of insulin secretory vesicles. Finally, once diabetes is established, chronic hyperglycaemia and hyperlipidaemia can exert deleterious effects on B-cell function, respectively referred to as glucotoxicity and lipotoxicity.It is conceivable that glucotoxicity and lipotoxicity interdependently converge toward the generation of damaging effectors on B-cell function *(Fonseca V et al 2010).*

2-3-3Pancreatitis

Pancreatitis is inflammation of the pancreas. The pancreas is a large organ behind the stomach that produces digestive enzymes. There are two main types, acute pancreatitis and chronic pancreatitis. Symptoms of pancreatitis include pain in the upper abdomen, nausea and vomiting. The pain often goes into the back and is usually severe. In acute pancreatitis a fever may occur and symptoms typically resolve in a few days. In chronic pancreatitis weight loss, fatty stool, and diarrhea may occur. Complications may include infection, bleeding, diabetes, or problems with other organs(*D. Whitcomb 2006)*

The most common causes of acute pancreatitis are gallstones and heavy alcohol use. Other causes include direct trauma, certain medications, infections such as mumps, and tumors among others. Chronic pancreatitis may develop as a result of acute pancreatitis. It is most commonly due to many years of heavy alcohol use. Other causes include high levels of blood fats, high blood calcium, some

medications, and certain genetic disorders such as cystic fibrosis among others.(*D. Whitcomb 2006).*

Smoking increases the risk of both acute and chronic pancreatitis, Diagnosis of acute pancreatitis is based on a threefold increase in the blood of either amylase or lipase. In chronic pancreatitis these tests may be normal. Medical imaging such as ultrasound and CT scan may also be useful. Acute pancreatitis is usually treated with intravenous fluids, pain medication, and sometimes antibiotics. Typically no eating or drinking is allowed and a tube may be placed into the stomach. A procedure known as a endoscopic retrograde cholangiopancreatography (ERCP) may be done to open the pancreatic duct if blocked. In those with gallstones the gallbladder is often also removed. In chronic pancreatitis, in addition to the above, temporary feeding through a nasogastric tube may be used to provide adequate nutrition. Long-term dietary changes and pancreatic enzyme replacement may be required. And occasionally surgery is done to remove parts of the pancreas.(*D. Whitcomb 2006).*

Acute pancreatitis occurs in about 30 per 100,000 people a year.New cases of chronic pancreatitis develop in about 8 per 100,000 people a year and currently affect about 50 per 100,000 people in the United States.Globally, in 2013 pancreatitis resulted in 123,000 deaths up from 83,000 deaths in 1990.It is more common in men than women. Often chronic pancreatitis starts between the ages of 30 and 40 while it is rare in children. Acute pancreatitis was first described on autopsy in 1882 while chronic pancreatitis was first described in 1946.(*D. Whitcomb 2006).*

2-3-4pancreatic cancer:

Pancreatic cancer arises when cells in the pancreas, a glandular organ behind the stomach, begin to multiply out of control and form a mass. These cancer cells have the ability to invade other parts of the body. There are a number of types of pancreatic cancer. The most common, pancreatic adenocarcinoma, accounts for about 85% of cases, and the term "pancreatic cancer" is sometimes used to refer only to that type. These adenocarcinomas start within the part of the pancreas which make digestive enzymes. Several other types of cancer, which collectively represent the majority of the non-adenocarcinomas, can also arise from these cells. One to two in every hundred cases of pancreatic cancer are neuroendocrine tumors, which arise from the hormone-producing cells of the pancreas. These are generally less aggressive than pancreatic adenocarcinoma.*(Christians KK 2014).*

Signs and symptoms of the most common form of pancreatic cancer may include yellow skin, abdominal or back pain, unexplained weight loss, light-colored stools, dark urine and loss of appetite. There are usually no symptoms in the disease's early stages, and symptoms that are specific enough to suspect pancreatic cancer typically do not develop until the disease has reached an advanced stage. By the time of diagnosis, pancreatic cancer has often spread to other parts of the body.*(Christians KK 2014).*

Pancreatic cancer rarely occurs before the age of 40, and more than half of cases of pancreatic adenocarcinoma occur in those over 70.Risk factors for pancreatic cancer include tobacco smoking, obesity, diabetes, and certain rare genetic conditions. About 25% of cases are linked to smoking, and 5–10% are linked to inherited genes. Pancreatic cancer is usually diagnosed by a combination of medical imaging techniques such as ultrasound or computed tomography, blood tests, and examination of tissue samples (biopsy. The disease is divided into stages,

from early (stage I) to late (stage IV). Screening the general population has not been found to be effective. *(Christians KK 2014).*

The risk of developing pancreatic cancer is lower among non-smokers, and people who maintain a healthy weight and limit their consumption of red or processed meat. A smoker's chance of developing the disease decreases if they stop smoking, and almost returns to that of the rest of the population after 20 years. Pancreatic cancer can be treated with surgery, radiotherapy, chemotherapy, palliative care, or a combination of these. Treatment options are partly based on the cancer stage. Surgery is the only treatment that can cure the disease; it may also be done to try to improve quality of life without the potential for cure. Pain management and medications to improve digestion are sometimes needed. Early palliative care is recommended even for those receiving treatment that aims for a cure*(Christians KK 2014).*

In 2012, pancreatic cancers of all types were the seventh most common cause of cancer deaths, resulting in 330,000 deaths globally. Pancreatic cancer is the fifth most common cause of death from cancer in the United Kingdom, and the fourth most common in the United States. The disease occurs most often in the developed world, where about 70% of the new cases in 2012 originated. Pancreatic adenocarcinoma typically has a very poor prognosis: after diagnosis, 25% of people survive one year and 5% live for five years. For cancers diagnosed early, the five-year survival rate rises to about 20%. Neuroendocrine cancers have better outcomes; at five years from diagnosis, 65% of those diagnosed are living, though survival varies considerably depending on the type of tumor. *(Christians KK 2014).*

2-3-5Pancreatic cysts

Pancreatic cysts are collections (pools) of fluid that can form within the head, body, and tail of the pancreas. Some pancreatic cysts are true cysts (non-

inflammatory cysts), that is, they are lined by a special layer of cells that are responsible for secreting fluid into the cysts. Other cysts are pseudocysts (inflammatory cysts) and do not contain specialized lining cells. Often these pseudocysts contain pancreatic digestive juices because they are connected to the pancreatic ducts. Pancreatic cysts can range in size from several millimeters to several centimeters. Many pancreatic cysts are small and benign and produce no symptoms, but some cysts become large and cause symptoms, and others are cancerous or precancerous. (Precancerous cysts are benign cysts that have the potential to become cancerous.).*(Christians KK 2014).*

Different types of cysts contain different types of fluids. For example, pseudocysts that form after an attack of acute pancreatitis contain digestive enzymes such as amylase in high concentrations. Mucinous cysts contain mucus (a proteinaceous liquid) produced by the mucinous cells that form the inside lining of the cyst. *(Christians KK 2014).*

The symptoms of pancreatic cysts depend on their size and location. Small (less than two cm) cysts usually cause no symptoms. Large pancreatic cysts can cause abdominal pain and back pain presumably by exerting pressure on the surrounding tissues and nerves.*(Christians KK 2014).*

Small or large cysts in the head of the pancreas also may cause jaundice (yellowing of the skin and eyes with darkening of the urine) due to obstruction of the common bile duct. (Obstruction causes bile to back up and forces bilirubin--the chemical that produces jaundice--back into the bloodstream and forces it to be excreted in the urine.) If the cysts become infected, it may result in fever, chills, and sepsis.On rare occasions, large pseudocysts can compress the stomach or the duodenum leading to obstruction to the movement of food in the intestines, resulting in abdominal pain and vomiting.*(Christians KK 2014).*

If a cyst becomes malignant and begins to invade the surrounding tissues, it may lead to the same type of pain as pancreatic cancer, pain that usually is constant and felt in the back and upper.*(Christians KK 2014).*

2-4-1 Ultrasound physics:

Ultrasound literally means “above or beyond sound”, that is, it is the sound above the human audible (hearing) range.Although ultrasound was first used on a large scale practical basis in World War I in order to detect the position and depth of submarines and was referred to as Sonar, it was not until 20 years later that it was first applied to medicine (sonography).the frequency of sound that is in the human audible range is between 15 Hz to 20 kHz (Note: 1 Hz = 1 Hertz = 1 cycle/second, 1 kHz = 1,000 Hz) ultrasound frequencies are higher than that of audible sound and thus comprises of sounds with frequencies greater than 20 kHz (similarly, sound under 20 kHz is called infrasound “below sound”) the range of sound that is commonly used in Radiology (Diagnostic Ultrasound)is between 1 MHz and 20 MHz (Note: 1 MHz = 1,000 kHz = 1,000,000 Hz).

Characteristics of Sound:

A sound beam is similar to an x-ray ray beam in that both are wave transmittingenergy. A more important difference is that x-rays pass readily through a vacuum while sound requires a medium for its transmission.

The velocity of sound depends on the nature of the medium.

Longitudinal Waves:

Ultrasound waves are transmitted through tissue as longitudinal waves of alternating compression and rarefaction regions.

the wave front starts at time 1 when the vibrating piston compresses the adjacent material a band of rarefaction is produced at time 2, when the piston reverses its direction

each repetition of this back-and-forth motion is called a cycle, and each cycle produces a new wave.

Wavelength:

The length of the wave (or wavelength) is the distance between two bands of compression or rarefaction and is represented by the symbol □ (has unit of millimeters).

Frequency:

The motion of the vibrating piston, plotted against time, forms the sinusoidal curve shown along the left side.

The frequency is the number of compressions (or rarefactions) bands that pass any given point in space per unit time and is measured in Hertz which is defined as:

$$1 \text{ Hertz} = 1 \text{ cycle per second}$$

frequency has the units of Hz (or 1/second or sec-1 or s-1)

Period:

The period is the time between compression or rarefaction (oscillations) bands and has the units of time (seconds). In other words, the period is the time that it takes for one cycle to occur.

The relationship between period and frequency is:

$$\text{frequency (Hz)} = \frac{1}{\text{period (s)}}$$

Physical Density:

Dense material tend to be composed of massive molecules, and these molecules have a great deal of inertia. They are difficult to move or to stop once they are moving.Because the propagation of sound involves the rhythmic starting and stopping ofparticulate motion, we would not expect a material made up of large molecules (i.e.: large in mass) such as mercury to transmit sound at as great a speed as a material composed of smaller molecules, such as water.the speed of sound in a material decreases if the density is increased, assuming

constant stiffness

Acoustic Impedance:

Acoustic impedance (Z) of a material is given by:

impedance (Rayl) = speed of sound (m/s) • density of material (kg/m3) in material.

the acoustic impedance unit is called the Rayl (kg/m2/s) acoustic impedance can be considered to be a measure of a material's ability to transmit acoustic energy (air and lung media have low values, and bone and metal have high values)

acoustic impedance is determined by the density and stiffness of a medium

since the speed of sound is independent of frequency in the diagnostic ultrasound range, acoustic impedance is also independent of frequency

acoustic impedance determines the amount of energy reflected at an interface

since the speed of sound in tissue is relatively constant in the diagnostic ultrasound range, then the acoustic impedance of most tissues is also a constant, they typically have values around 1.6 x 106 kg/m2/s (Rayls)

Intensity:

The intensity (or loudness) of sound is determined by the length of oscillation of the particles (vibration amplitude) conducting the waves.the harder the piston is struck, the more energy it receives and the wider its vibration

Amplitude these wider excursions are transmitted to the adjacent conducting media and produce a more intense beam in time the vibrations diminish in intensity although not in frequency, and the sound intensity decreases, producing a lower intensity beam ultrasound intensities are expressed in power per unit area, where power (mW) is the rate at which acoustic energy is transferred and the area (cm2) > the ultrasound beam at some distance from the transducer surface, thus:

$$\text{intensity (mW/cm2)} = \frac{\text{power (mW)}}{\text{area (cm2)}}$$

Interaction of Ultrasound with Matter:

Absorption:

The term “absorption” refers to the conversion of ultrasonic energy to thermal energy, and is the dominate contribution to attenuation in soft tissue.The mechanisms involved in absorption are rather complex (and thus will not be discussed in detail); the three primary factors that determine the amount of absorption are:

(1) the viscosity of the conducting medium

(2) the “relaxation time” of the medium; and

(3) the frequency of the sound wave Viscosity

viscosity is an internal friction (or a frictional force) that opposes the motion of the particles in the medium particle freedom decreases and internal friction increases with increasing viscosity

this internal friction absorbs the sound, or decreases its intensity, by converting sound into heat in liquids, which have low viscosity, very little absorption takes place, in soft tissue viscosity is higher and a medium amount of absorption occurs, while bone shows high absorption of ultrasound.

Reflection:

A portion of the ultrasound beam is reflected at tissue interfaces as shown below. The sound reflected back towards the source is called a echo and is used to generate the ultrasound image.the percentage of ultrasound intensity reflected depends, in part, on the angle of incidenceas the angle of incidence increases, reflected sound is less likely to reach the transducer and thus no acoustic signal is received to image no reflection is generally detected by the transducer, if the angle of incidence is greater than 3° specular (smooth) reflection occurs from large, smooth surfaces (major contributor to ultrasound images)

Refraction:

Refraction is the change in direction of an ultrasound beam when passing through one medium to another.

When ultrasound passes from one medium to another, the frequency remains the Same but the wavelength changes to accommodate the new velocity of sound in the second medium (for diagnostic ultrasound, the speed of sound is independent of frequency).

Producing an image:

Probe emits a sound wave pulse-measures the time from emission to return of the echo Wave travels by displacing matter, expanding and compressing adjacent tissues It generates an ultrasonic wave that is propagated, impeded, reflected, refracted, or attenuated by the tissues it encounters.

Piezoelectric Effect:

Appearance of an electric potential across certain faces of a crystal when it is subjected to mechanical pressure.

The word originates from the greek word “piezein”, which means “to press”

Discovered in 1880 by Pierre Curie in quartz crystals.

Conversely, when an electric field is applied to one of the faces of the crystal it undergoes mechanical distortion.

Examples --- Quartz, Barium titanate, tourmaline.

2-4-2Ultrasound scanners:

-Transmitter.

-Transducer.

-Receiver.

-Processor.

-Display.

-Storage.

Transmitter:

a crystal makes energy into sound waves and then receives sound waves and converts to energy. This is the Piezoelectric effect

u/s machines use time elapsed with a presumed velocity (1,540 m/s) to calculate depth of tissue interface Image accuracy is therefore dependent on accuracy of the presumed velocity. A crystal makes energy into sound waves and then receives sound waves and converts to energy this is the piezoelectric effect

u/s machines use time elapsed with a presumed velocity (1,540 m/s) to calculate depth of tissue interface .Image accuracy is therefore dependent on accuracy of the presumed velocity.

A crystal makes energy into sound waves and then receives sound waves and converts to energy. This is the Piezoelectric effect U/S machines use time elapsed with a presumed velocity (1,540 m/s) to calculate depth of tissue interface

Image accuracy is therefore dependent on accuracy of the presumed velocity.

Transducers:

Continuous mode

continuous alternating current 34have 2 crystals –1 talks, 1 listens

Pulsed mode

Most used in Diagnostic U/S and the Crystal talks and then listens

Receiver:

Sound waves hit and make voltage across the crystal- The receiver detects and amplifies these voltages Compensates for attenuation

Displays:

-B-mode

-Real time gray scale, 2D

-Flip book- 15-60 images per second

-M-mode

-Echo amplitude and position of moving targets Valves, vessels, chambers.

Image Resolution:

Axial Resolution:

Ability to differentiate two objects along the long axis of the ultrasound beam

Determined by the pulse length Product of wavelength λ and # of cycles in pulse

Decreases as frequency f increases Higher frequencies produce better resolution.

Lateral Resolution:

The ultrasound beam is made up of multiple individual beams The individual beams are fused to appear as one beam.

The distances between the single beams determines the lateral resolution.

Ability to differentiate objects along an axis perpendicular to the ultrasound beam

Dependent on the width of the ultrasound beam, which can be controlled by focusing the beam Dependent on the distance between the objects.

2-4-3Artifacts:

Reverberation Artifact:

In reverberation artifacts, the sound bounces back and forth between two interfaces. This prolongs the time of flight, producing an artifact deep to the interface. In this situation, some of the sound returning to the transducer is reflected back into the patient. That pulse strikes the same interface in the patient and is reflected back to the transducer a second time. The first reverberation artifact is, therefore, twice as far from the skin surface as the original interface was from the transducer

This phenomenon often occurs when the sound beam is perpendicular to a strong reflector, such as a soft tissue–air interface or the abdominal wall deep to a considerable depth of subcutaneous adipose tissue. Depending on the intensity of the reflection of the interface and the degree of echogenicity of the tissue deep to the interface, a second and even third reverberation artifact can occur. All of these are spaced equal to the distance from the transducer to the actual interface.

Generally, the reverberation artifacts caused by the abdominal wall do not cause confusion. If there is a fluid collection deep to the abdominal wall, such as the bladder, there should be no difficulty appreciating the artifactual nature of the echoes. There can be real echoes from sludge in the dependent portion of fluid collections. Echoes in the superficial (nondependent) aspect of the bladder are easily appreciated as artifacts. On the other hand, reverberation artifacts can be superimposed over the superficial portion of the liver. This gives the appearance of increased echogenicity.

Although the TGC may be manipulated to balance the echogenicity in the near and far portions of the liver, the near echo may not represent true interfaces in the liver. When watching during real-time scanning, the true echoes can be seen to move with respiration, whereas the artifactual echoes appear like a haze through which the liver is being viewed. These artifacts, however, can easily obscure superficial metastases and superficial cysts

If the artifactual echo appears in the right (or wrong) place, it can cause diagnostic difficulty. For example, a first reverberation artifact could be superimposed over the lateral aspect of the kidney to simulate a subcapsular hematoma .Once again, watching the kidney move during real-time scanning and moving the plane of section of the transducer should help clarify the issue. The hard-copy image, however, can be confusing.

Ultrasound experts usually have had the experience of being mistaken, or at least perplexed, by an artifactual fluid collection simulating a real pelvic cyst or abscess. In this case, the combination of two artifacts simulates the fluid collection. The reverberation is generally caused by air within the rectum or other loop of bowel. The air not only reflects the sound back to cause the reverberation artifact but also causes distal acoustic shadowing, resulting in an echo-free shadow. The reverberation artifact is then superimposed on the echo-free shadow to simulate the deep wall of the fluid collection. In this situation, the first step is to consider the possibility that the fluid collection in the pelvis could be an artifact. Attempting different angles of the plane of section from different windows on the abdominal wall may be helpful. Partially emptying the bladder can show that the artifactual fluid collection also decreases in size.

This is because the difference from the transducer to the air interface deep to the bladder decreases, and therefore, the distance from the air interface to the

artifactual wall also decreases. It is important not to empty the bladder completely lest gas-filled bowel interposes, hiding a real fluid collection.

A mirror-image artifact (see next section) can cause a similar appearance, so that the depth of the deep wall of the cyst may not be the same as the distance from the transducer to the rectal air.

Mirror Magnification:

The mirror-image artifact is similar to the reverberation artifact. In a reverberation artifact, the sound pulse is reflected back into the body from the transducer–skin interface. In the mirror-image artifact, the extra reflection comes from within the body. Although the extra reflection may be within the line of the sound beam, more commonly, the sound is reflected off an angle to another interface so that, like a real mirror, the artifact shows up as the virtual object. An example of this situation is a hemangioma in the liver that appears to be a lesion within the lung above the diaphragm

. The sound is reflected off the diaphragm back into the liver. Because the angle of reflection is equal to the angle of incidence, the sound pulse then hits the interfaces within the hemangioma. The sound pulse is then reflected back to the diaphragm once again, with an angle of reflection equal to the angle of incidence, and then back to the transducer. The machine straightens the path of the returning echo, assuming that the interfaces were coming from the direction in which transducer was pointing and at a distance corresponding to the actual time of flight.

As mentioned earlier, the mirror-image artifact may also simulate a pelvic fluid collection similar to the reverberation artifact .

The sound may be reflected off the rectal air at an angle so that the deep wall of the artifactual cyst represents the mirror image of the inferior and anterior walls of the bladder. In this case, it is not helpful to measure the distances from the

transducer. Especially in the transverse image, only a short segment of the bladder is traversed, yet the fluid collection may appear large. This is because the sound is being reflected out of the plane of section to hit the inferior wall of the bladder.

Mirror-image artifacts can cause other strange appearances, such as invasion of a transitional cell carcinoma through the bladder wall (Fig. 2-8). An empyema or lung abscess can be simulated by a mirror-image artifact of a hepatic cyst .

Ring down Artifacts:

The ring-down artifact, also known as the comet tail artifact, appears as a line in the direction of the sound beam and deep to a strong reflector.4–6 The cause can be a piece of metal in the body, such as a surgical clip or lead shot. More commonly, the ring-down artifact is seen deep to a collection of gas. In fact, the artifact is not an echogenic line but rather a collection of closely spaced perpendicular echoes along one or two vectors in the ultrasound image. Originally, these were thought to be sound entering the metal or gas bubble and reverberating back and forth within the structure, each time sending some of the sound back to the transducer. This explanation would account for a series of closely spaced echoes equal to the depth of the piece of metal or gas bubble. A more plausible explanation, however, is equivalent to the ringing of a bell. The sound pulse insonates the metal, causing it to ring. Interestingly, the artifact does not occur deep to calcification or calculi.

With gas bubbles, there is a slightly different explanation. Not all collections of gas produce ring-down artifacts, but the ring-down artifact can be characteristic of certain configurations of gas bubbles. The “bugle” of fluid trapped between four small bubbles (the bubble tetrahedron) is the source of the artifact.7 The sound pulse insonates the bugle, causing it to vibrate and send a prolonged sound wave back to the transducer.

A variant of this artifact occurs deep to cholesterol crystals, usually in the wall of the gallbladder. This has been referred to as the V-shaped artifact.8 These artifacts have only two or three rings, and the more distal ones are always smaller than the proximal ones. The platelike cholesterol crystals probably are oriented perpendicular to the beam to cause the ringing.

Shadowing and Enhancement:

The intensity of an echo is determined not only by the strength of the reflection but also by the acoustic characteristics of the tissue between the transducer and the interface. The sound travels through the intervening tissue on the way down to the interface, and the returning echo must also traverse the same tissue. The intervening tissue may attenuate or appear to enhance the intensity of the returning echo.

Enhancement Through Transmission

An increase in the amplitude of the echoes deep to a structure is called enhancement through transmission.. This is a characteristic of cysts but is actually a misnomer. The fluid only attenuates the sound less than the surrounding tissue. The cystic fluid causes almost no attenuation of the sound. Adjacent to the cyst, the liver parenchyma, for example, does cause some attenuation. This is compensated by the TGC, which makes the liver echoes uniform from superficial to deep. The TGC overcompensates through the cyst, causing the deeper echoes to appear brighter . The value of this artifact is that it greatly increases confidence in the diagnosis of a cyst.

Sometimes, a cyst is too small for the enhancement to be explained only by the absence of attenuation. In fact, the small cyst can act as a lens, refocusing the sound beam, similar to using a magnifying glass to focus the sun’s rays to burn a hole in a dead leaf. The refocusing of the sound beam also causes enhancement through transmission.

Acoustic Shadowing

When there is acoustic shadowing (e.g., deep to a gallstone), the sound pulse does not reach the deeper tissues to produce an echo. Complete shadowing does not cause confusion and can be helpful in confirming a gallstone or a renal stone. In fact, the ultrasonographer should be wary of making the diagnosis of a calculus without its tell-tale shadow. In some situations, a shadow may not be complete deep to a calculus. This is due to the width of the beam relative to the diameter of the calculus. When the calculus is smaller than the beam, an echo is received from the calculus, but some of the sound goes around the stone, with echoes returning from the deeper structures. There may be no shadow at all. It is important to use the appropriate transducer and to adjust the transmit focus to the depth of the stone to appreciate the shadow. Focusing plays an important role in the production of acoustic shadows.

In clinical practice, it is important to differentiate shadows due to gas-filled structures from calcified objects. Hard or calcified structures, including bone, reflect about 30% of the sound and absorb the rest; thus, the shadows are relatively "clean." Gas collections reflect about 99% of the sound energy, so diffuse reverberations can fill the shadow with noise, making it relatively "dirty." For example, it is important to differentiate gas in the biliary tree (pneumobilia) from calculi. In addition to the different characteristics of the shadows, the gas bubbles tend to have caps from short off-axis artifacts, whereas the gallstones have curved proximal surfaces.

It is more difficult to appreciate the artifactual nature of relative shadowing. For example, if the sound beam is attenuated but not completely blocked, an area of the liver may look echo poor and simulate a metastasis or focal sparing of fatty infiltration.

Acoustic shadowing can also occur deep to oblique interfaces owing to refraction. Refraction has already been discussed in terms of deflecting the whole of the sound beam to interfaces deep to the edge of a cyst. The edge of a cyst, however, can also refract parts of the sound beam away from the other parts not traversing the edge of the cyst. The effect is defocusing. A defocused beam has less intense sound, less intense returning echoes, and therefore a relative shadow the refractive shadowing can also cause confusion if it is more diffuse and only relatively attenuating. This frequently occurs through the lower uterine segment owing to the oblique interface of the superior aspect of the bladder

. In the liver, the fissure for the ligamentumvenosum may contain some fat, and the oblique interface can cause relative shadowing of the caudate lobe, simulating a metastasis.19 Portions of the pancreas can appear echo poor owing to interposed fat deep to the left lobe of the liver. A relatively uncommon but interesting artifact is the two-tone testis artifact, caused by refraction of the testicular artery20 .

Beam Width Artifact:

Although the sound beam is displayed on the image as a narrow vector, the beam does have a finite width. It is at least several millimeters wide in the focal zone and even wider in the near and far zones. The intensity is higher in the center of the beam, decreasing toward the periphery. The periphery of the sound beam can be reflected by adjacent structures. When the center of the beam is aimed beside a strong reflector, an off-axis echo can be displayed along the vector representing the center of the beam. Side lobes of the main beam can cause echoes to appear from structures as much as 45 degrees away from the central vector.

The same artifact can be detected when the plane of section is aimed away from the strong reflector. In this case, the cause of the artifact is less apparent. Side-lobe artifacts are much more likely to occur with phased array and curved linear array transducers. Fortunately, they are usually easily differentiated from real septations.

This same mechanism can also cause low-level echoes within cystic structures, mimicking sludge, pus, or debris; in these situations, it is called beam-width or slice-thickness artifact. Beam-width artifact is a partial volume effect and is analogous to the artifact described for computed tomography. Its occurrence can be diminished using the correct transmit focal zone, making the slice as thin as possible.

Side-lobe and grating-lobe artifacts can be diminished using the same maneuvers described for beam-width artifacts because they are also dependent on the incident angle and are not dependent on gravity. Furthermore, grating lobes can be avoided when apodisation is used in the transducer. In this process, the lateral transducer elements have less energy than central elements. Another process, called spatial filtering, makes the echoes from the peripheral transducer elements less amplified, again making grating lobes less likely to occur. Dynamic focusing of the returning echo likewise tends to enhance echoes along the central axis and diminish the effects of off-axis interfaces.

Aside from the beam-width and side-lobe artifacts, a third artifact can induce the presence of low-level echoes in cystic structures. This is the range-ambiguity artifact.23 These echoes are related to the use of fast frame rates and high pulse repetition frequencies. When the depth setting is low, as for superficial structures, echoes from deeper interfaces may return to the transducer after the next pulse has been fired. These echoes are interpreted as an interface much closer to the transducer. Usually, these are not noted because they are low-intensity echoes that are lost in echogenic tissue.

A large cystic structure (e.g., on an endovaginal scan), however, can cause these echoes to be misinterpreted as representing the far wall of the cyst. For objects situated deep in the body, or even for the dorsal skin, the time of the returning echoes can be long, and they can reach the transducer just after a second pulse is

emitted. The machine interprets these echoes as originating in superficial structures. They can also be second reverberation echoes. They are only noted if they are superimposed over cystic structures (e.g., gall bladder, bladder, cysts).

2-5-1 Ultrasound techniques:

Because the pancreas lies posterior to the stomachand duodenum, a variety of techniques must usuallybe employed to examine it fully. Although ultrasoundmay still be considered the first line of investigation,CT, MRI and/or endoscopic retrogradecholangiopancreatography (ERCP) are frequentlyrequired to augment and refine the diagnosis.*(Jane Bates ,Abdominal Ultrasound, second Edition2004)*

The operator must make the best use of availableacoustic windows and different patient positionsand techniques to investigate the pancreas fully.The most useful technique is to start by scanningthe epigastrium in transverse plane, using theleft lobe of the liver as an acoustic window. Usingthe splenic vein as an anatomical marker, the bodyof the pancreas can be identified anterior to this. The tail of pancreas is slightly cephalic to the head,so the transducer should be oblique accordinglyto display the whole organ..*(Jane Bates ,Abdominal Ultrasound, second Edition2004)*

Different transducer angulations display differentsections of the pancreas to best effect:

- Identify the echo-free splenic vein and thesuperior mesenteric artery posterior to it. Thelatter is surrounded by an easily visible,hyperechoic fibrous sheath. The pancreas is‘draped’ over the splenic vein.
- Where possible, use the left lobe of the liver asan acoustic window to the pancreas, anglingslightly caudally.
- The tail, which is often quite bulky, may require the transducer to be angled towards the patient’s left. The spleen also makes a good window to the tail in coronal section. If you can’t see the pancreatic head properly, turn the patient left

side raised, which moves the duodenal gas up towards the tail of the pancreas. Right side raised may demonstrate the tail better. If these maneuvers still fail to demonstrateThe organ fully, try:

—asking the patient to perform the Valsalva manoeuvre with abdominal protrusion

—scanning the patient erect

—filling the stomach with a water load to create an acoustic window through which the pancreas can be seen.*(Jane Bates ,Abdominal Ultrasound, second Edition2004)*

2-5-2Ultrasound appearances:

The texture of the pancreas is rather coarser than that of the liver. The echogenicity of the normal pancreas alters according to age. In a child or young person it may be quite bulky and relatively hypoechoic when compared to the liver. In adulthood, the pancreas is hyperechoic compared to normal liver, becoming increasingly so in the elderly, and tending to atrophy. (*Jane Bates ,Abdominal Ultrasound, second Edition2004)*

The pancreas does not have a capsule and its margins can appear rather ill-defined, becoming infiltrated with fat in later life.

These age-related changes are highly significant to the sonographer; what may be considered normal in an elderly person would be abnormally hyperechoic in a younger one, and may represent a chronic inflammatory state. Conversely a hypoechoicapancreas in an older patient may represent acute inflammation, whereas the appearances would be normal in a young person. *(Jane Bates ,Abdominal Ultrasound, second Edition2004)*

The main pancreatic duct can usually be visualized in the body of pancreas, where its walls are perpendicular to the beam. The normal diameter is 2 mm or less.The common bile duct can be seen in the lateral portion of the head and the

gastroduodenal artery liesanterolaterally. The size of the uncinate process varies.*(Jane Bates ,Abdominal Ultrasound, second Edition2004).*

2-5-3Pitfalls in scanning the pancreas:

The normal stomach or duodenum can mimic pancreatic pathology if the patient is insufficientlyfasted. A fluid-filled stomach can be particularly difficult when looking for pancreatic pseudocysts in patients with acute pancreatitis. Giving the patient a drink of water usually differentiates the gastrointestinal tract from a collection. Epigastric or portal lymphadenopathy may alsomimic a pancreatic mass. If careful scanning and appropriate patient positioning are unable to elucidate, CT is normally the next step. (*Jane Bates ,Abdominal Ultrasound, second Edition2004)*

2.6. Previous studies:

M Gouldet al**;** 1985studied size of pancreas in diabetic patient based ultrasound. and this study in UK using a real time linear array system (Picker LS 3000), they tested Sixty adult diabetic patients: 22 had insulin dependent diabetes (group 1) and 19 non-insulin dependent diabetes (group 2) and 19were non-ketotic patients who had to be given insulin because of inadequacyof diabetic control with oral hypoglycaemic agents (group 3). Nineteen healthycontrols were also studied.in this study measure the head(area medialy to SMA) and body(area anterior to SV) of pancreas, These results show that the pancreas is significantly smaller in diabetic patients than in healthy controls. Furthermore, patients with insulin dependent diabetes have significantly smaller pancreases than patients with non-insulin dependent disease. Non-ketotic patients whose diabetes was not controlled with maximum doses of oral hypoglycaemic agents and who required insulin had pancreases intermediate in size between those in the other two groups. This is the first study to document these changes systematically. The pattern of diminution in the size of the pancreas in diabetes parallels the impairment of exocrine function previously described by us. Thus patients with insulin dependent diabetes have the lowest serum concentrations of pancreatic enzymes. Patients with non-insulin dependent disease have marginally reduced serum pancreatic enzyme values, while patients with non-ketotic disease who require insulin because of inadequate control with oral hypoglycaemic agents have serum pancreatic enzyme values between those of the other two groups. In group 3 the sizes of the head and body of the pancreas were intermediate between those in groups 1 and 2.There was no correlation between size of the pancreas and body weight or duration of diabetes. *(M Gould et al; 1985).*

Another study byAlzaid A etal 2006 they studied evaluation of pancreas in diabetic by ultrasound,This study was in USA and the method was tested on 57 diabetic patients: 14 with Type 1 (insulin-dependent) diabetes, 10 insulin-treated and 33 tablet-treated patients with Type 2 (non-insulin-dependent) diabetes, and 19 non-diabetic subjects.in this study measure the head(area medially to SMA) and body(area anterior to SV) of pancreas.The result of there study ,The pancreas of patients with Type 1 diabetes was markedly smaller ($p < 0.0001$) than the pancreas in non-diabetic subjects. The pancreas of patients with Type 2 diabetes was more moderate in size: larger ($p < 0.001$) than that of Type 1 diabetic patients but smaller ($p < 0.5$) than the pancreas of the control group. Pancreatic size of patients with Type 2 diabetes was also related to basal insulin secretion with insulin-deficient patients (low or undetectable C-peptide) having smaller ($p < 0.05$) pancreases than those with normal insulin secretion. There was no difference in the size of the pancreas in the different treatment groups of Type 2 diabetic patients. Pancreatic size did not correlate with age, body mass index or the duration of diabetes. We conclude that the pancreas is a smaller organ in patients with diabetes mellitus and that the decrement in size is maximal in insulin-dependent/insulin-deficient subjects. Ultrasonography, therefore, can potentially serve to discriminate between the different types of diabetes(Alzaid A et2006).

And AfraaSiddig ,Caroline Edward and et al 2014 studied Characterization of pancreas in Sudanese PopulationUsing Computerized Tomography ,A total number of 241 Sudanese subjects were included in thestudy, 161 (66.8%) were males and 80 (33.2%) were females, their mean ages were 40.6 ± 16.1; all were examined usingcross-sectional computerized tomography (CT) imaging for abdomen. The subject's ages and gender were recorded and thebody characteristics including height, weight, BMI, abdomen circumference (AC), were evaluated and correlated

withpancreas size and CT number. This study revealed that the head of pancreas size was 27.9 ± 4.5 mm, the body was 23.1 ±3.7mm, and tail was 19.0 ± 3.1mm, while the CT number (Hounsfield unit) was59.1 ± 14, 57.3 ± 12.6 and 55.2 ± 13.1respectively. Also the study showed a significant relation between the pancreas size, pancreas CT number, age, and AC at($p<0.05$). they study The measurements were taken from the operator councilof the CT machine; the axial images were obtained throughthe middle of the pancreatic portion (head, body andtail) .Anterior-posterior diameters (AP) were measured at right angles to the longitudinal axis of the organ. The largestdiameter of the pancreas lying to the left of the middle of the Vertebral body was considered the head .The body of thepancreas was measured on the left margin of the vertebralbody and the tail opposite to the medial margin of the leftkidney. The transverse diameter of the adjacent vertebralbody was measured and used as a reference and marker ofbody character as applied by Andreas . The CT numbersfor the pancreas head, body and tail were measured(Hounsfield). The CT number of the lumber Vertebra wasalso been evaluated.They founded the concluded that axial CT scan is considered a an appreciable radiological method for measuring thepancreas size and characterizing its structure using CTnumber (Hounsfield). The study also revealed that theSudanese pancreas is different from what was mentioned inthe literature and other previous studies, and also pancreas size and CT number had significant relations with age andother body habitus. Sudanese pancreas size and CT numberwere best described by the established formulae for age, weight, height, AC, VBTD, VDCT. Local references forSudanese pancreas measurements and CT number wereestablished.*(AfraaSiddig , Caroline Edward and et al 2014)*

Chapter three

Materials and methods

3-1materials:

3-1-1Area and study and duration:

It was conducted in Khartoum- Sudan, Noreen diabetic center, during the period from June up to October 2015.

3-1-2 population of study:

Thirty fife adult diabetic patients were investigated (Ranges between 20-75), andthirty fife healthy controls were also studied with different ages (Ranges between (25-50 years old) were selected consecutively on this study. This study include all the diabetic patients with diabetic disease only, and exclude all diabetic patients with another disease.

3-1-3machine used:

A real time convex array system (3.75 MHz electronic transducer –Mindary DP-2200 ,china).

Features :

- Digital beam-former
- 10" non-interlaced monitor
- Wide applications: abdominal, urology, GYN, OB, small parts, and orthopaedics
- One USB port and optional DICOM
- Triple-frequency transducer series, max frequency up to 10MHz

- Two transducer connectors (optional)

Standard Configurations :

- DP-2200 main unit
- 10″non-interlaced monitor
- One transducer connector
- 128-frame CINE loop
- 90-frame image storage
- One USB port
- Measurement & calculation software packages
- Electronic convex array transducer: 35C50EB (2.5/3.5/5.0MHz)

Optional :

- Electronic linear array transducer: 75L38EB (5.0/7.5/10MHz)
- Electronic linear array transducer: 75L60EA (5.0/7.5/10MHz)
- Electronic endocavity transducer: 65EC10EB (5.0/6.5/8.0MHz)
- Electronic micro-convex array transducer: 65C15EA (5.0/6.5/8.0MHz)
- Electronic micro-convex array transducer: 35C20EA(2.5/3.5/6.0MHz)
- Two transducer connectors
- Needle-guided brackets
- DICOM3.0

- Footswitch
- Mobile trolley
- Hand carried bag

3-1-4 Datacollecting sheet:

Data collecting sheet is use to collect the date from number of patient.

3-2-Methods:

3-2- technique used:

The patients and controls were scanned in the morning after an overnight fast. Scans were performed with the patients supine and erect. A morecomplete and clearer visualization of the pancreas was achieved by scanningwith the patient erect. The head (defined as the area medial to the superiormesenteric vein) and the body of the pancreas were measured separately, since these were often visualized to best advantage in different views.As the head is often oriented in the longitudinal plane parallel to the inferiorvena cava, measurements were made in this plane below the portal veins well as in the transverse or oblique plane (taking the midpoint of the confluenceof the superior mesenteric and splenic veins as the marker point).In normal subjects the longitudinal section of the head was frequentlylarger than the transverse sections. This did not occur in any of the diabeticpatients. The tail of the pancreas was well seen as it passed anterior to the leftkidney, but the more distal portion extending into the splenic hilum (whichrepresents a very small part of the pancreatic mass) was rarely seen.The scans were recorded on photographic paper.

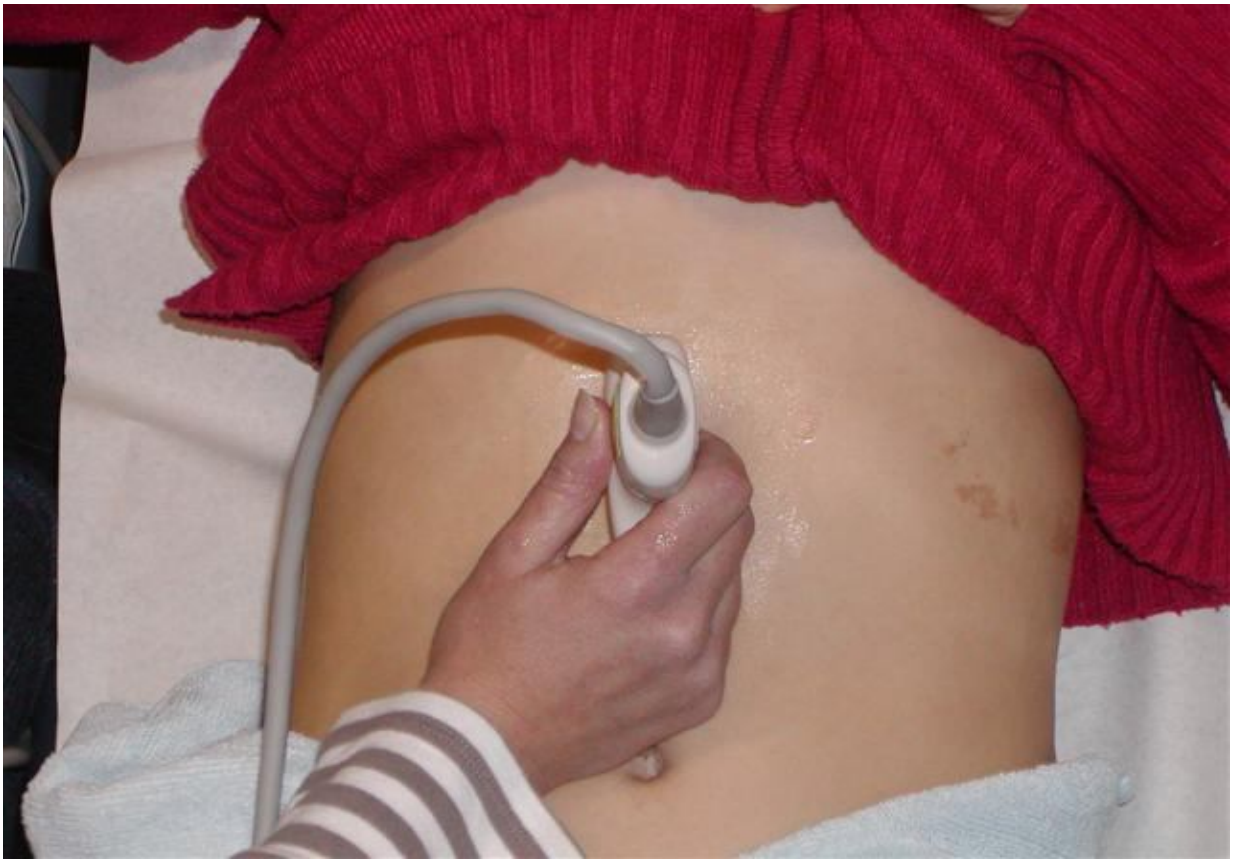


Figure (3-1) Ultrasoundof pancreas, longscan.

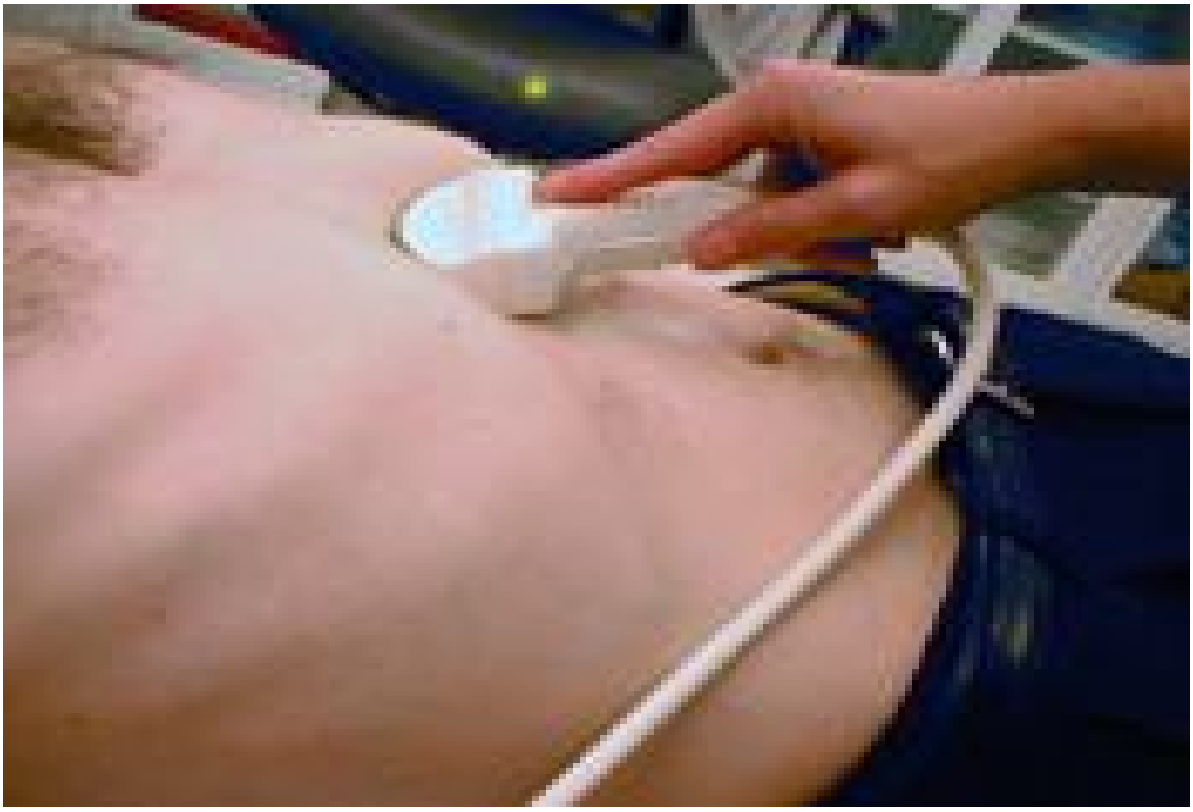


Figure (3-2) Ultrasoundof pancreas ,transeversescan.

Figure (3-3) Ultrasound machine (Mindary DP-2200)

3-2-2 Data collection:

The data sheet include:

Diabetic patients sheet:

-patient age.

-patient gender.

-type of diabetic.

-duration of diabetic.

-pancreatic measurements(head, body and tail).

Control Group sheet:

Diabetic patients sheet:

-patient age.

-patient gender..

-pancreatic measurements (head, body and tail).

3-2-3 Data analysis:

Microsoft Excel and SPSS program version 16 were used to analyze the data ofthis study. T- Test and F-test was used to examine the correlation between the variables,the correlation is significant at p value <0.05.

Chapter four

The results

Tables (4.1) shows the Mean ± Std. Deviation for patient related variables.

Descriptive Statistics	
Variable	**Mean ± Std. Deviation**
Age of the patient (yrs.)	35.54±11.843
Durationof Diabetic(yrs.)	7.957±5.5458
Ageof control group (yrs.)	29.40±8.605

Table (4.2) shows the Mean± Std. deviation for pancreatic measurement in control group

Variables	Mean± STD normal control group
Head of pancreas	2.2400±0.02981
Body of pancreas	2.1550±0.16325
Tail of pancreas	2.1690±0.13218

Table (4.3) shows the Mean± Std. deviation for pancreatic measurement in diabetic patients

Variables	Mean± STD diabetic patient
Head of pancreas	1.7689±0.46944
Body of pancreas	1.7057±0.45546
Tail of pancreas	1.7454±0.46682

Table (4-4) shows the age of Diabetic Patients.

The Age range	Frequency	Percent
20-30	16	45.7
30-40	10	28.6
40-50	5	14.3
50-60	3	8.6
60-70	1	2.9
Total	35	100.0

Table(4-5)shows the age of (healthy patients)control group

The age range	Frequency	Percent
20-25	8	22.8
25-30	5	14.2
30-35	5	14.2
35-40	5	14.2
40-45	4	11.4
45-50	8	22.8
Total	35	100.0

Table(4-6)shows Paired Samples Test

Paired Samples Test								
Pairs/ variables	Paired Differences					T	df	Sig. (2-tailed)
	Mean	Std. D	Std. Error Mean	95% Confidence Interval of the Difference				
				Lower	Upper			
Normal head – head (cm)	0.714	0.5003	0.1582	0.3561	1.0719	4.513	9	0.001
Normal body – body (cm)	0.735	0.4437	0.1403	0.4176	1.0524	5.239	9	0.001
Normal tail–tail (cm)	0.722	0.4854	0.1535	0.3749	1.069	4.704	9	0.001

Table(4-7) shows Independent Samples Test.

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	Df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval	
									Lower	Uppe
headcm	Equal variances assumed	71.639	.000	-4.516	53	.000	-.47666986	.10554897	-.68837428	-.264
	Equal variances not assumed			-5.979	34.631	.000	-.47666986	.07971754	-.63856678	-.314
bodycm	Equal variances assumed	31.095	.000	-4.244	53	.000	-.44929	.10586	-.66162	-.236
	Equal variances not assumed			-5.273	46.778	.000	-.44929	.08520	-.62072	-.277
tailcm	Equal variances assumed	41.532	.000	-3.954	53	.000	-.42357	.10713	-.63844	-.208
	Equal variances not assumed			-5.027	42.706	.000	-.42357	.08426	-.59353	-.253

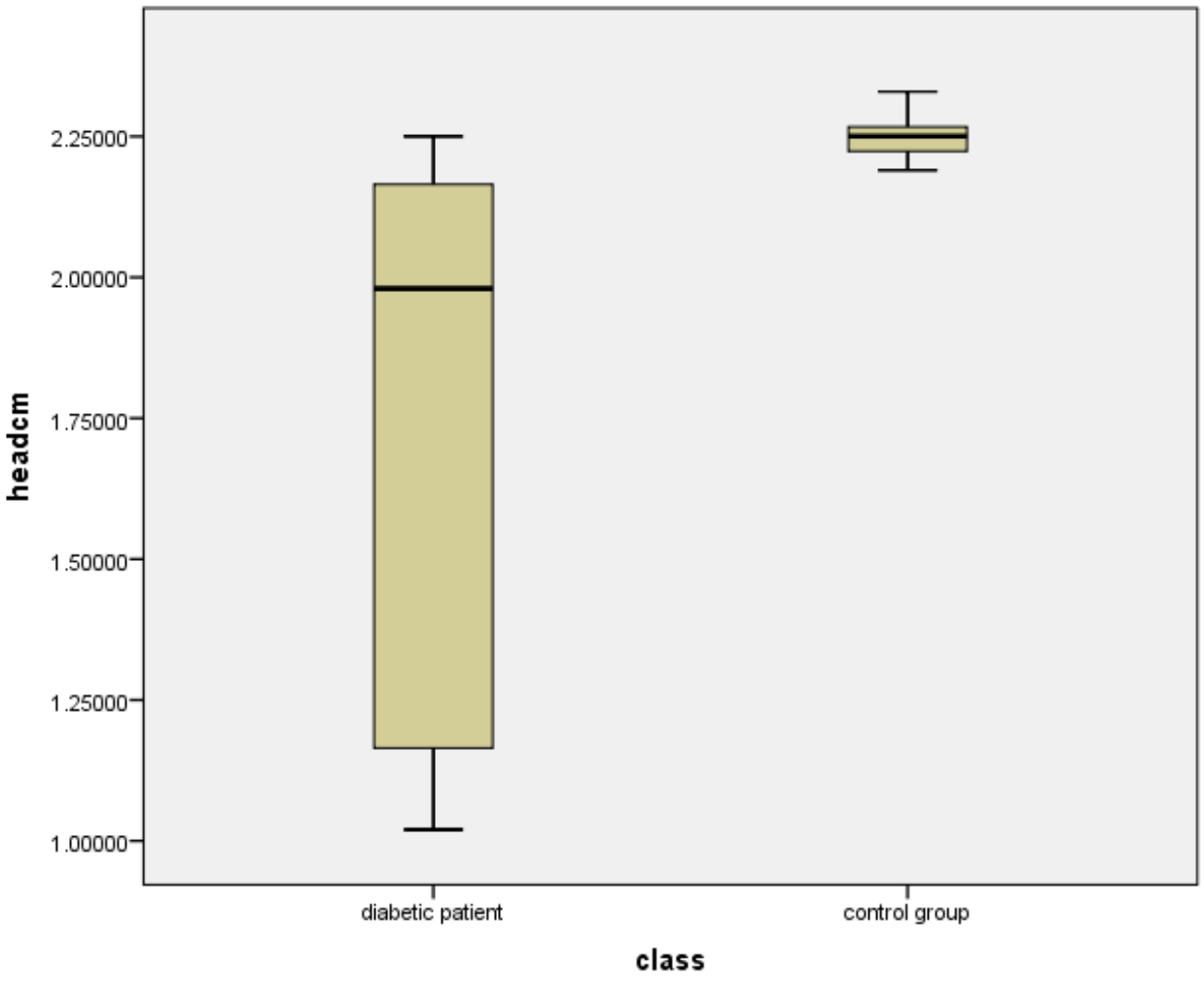
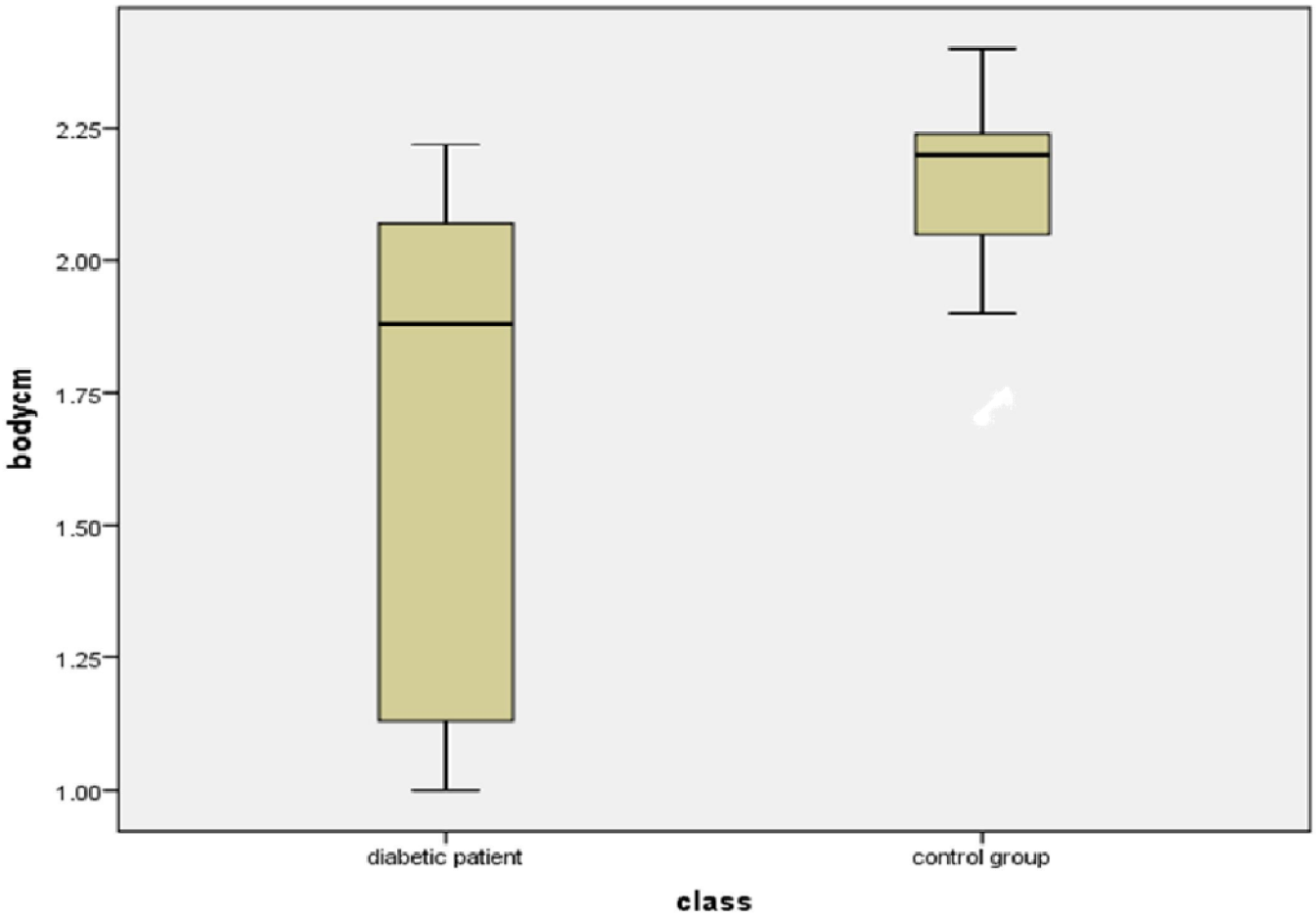


Figure (4.1) boxplot shows measure the head of pancreas in control and diabetic group.



Figure(4.2)boxplot shows measure the body of pancreas in control and diabetic groups

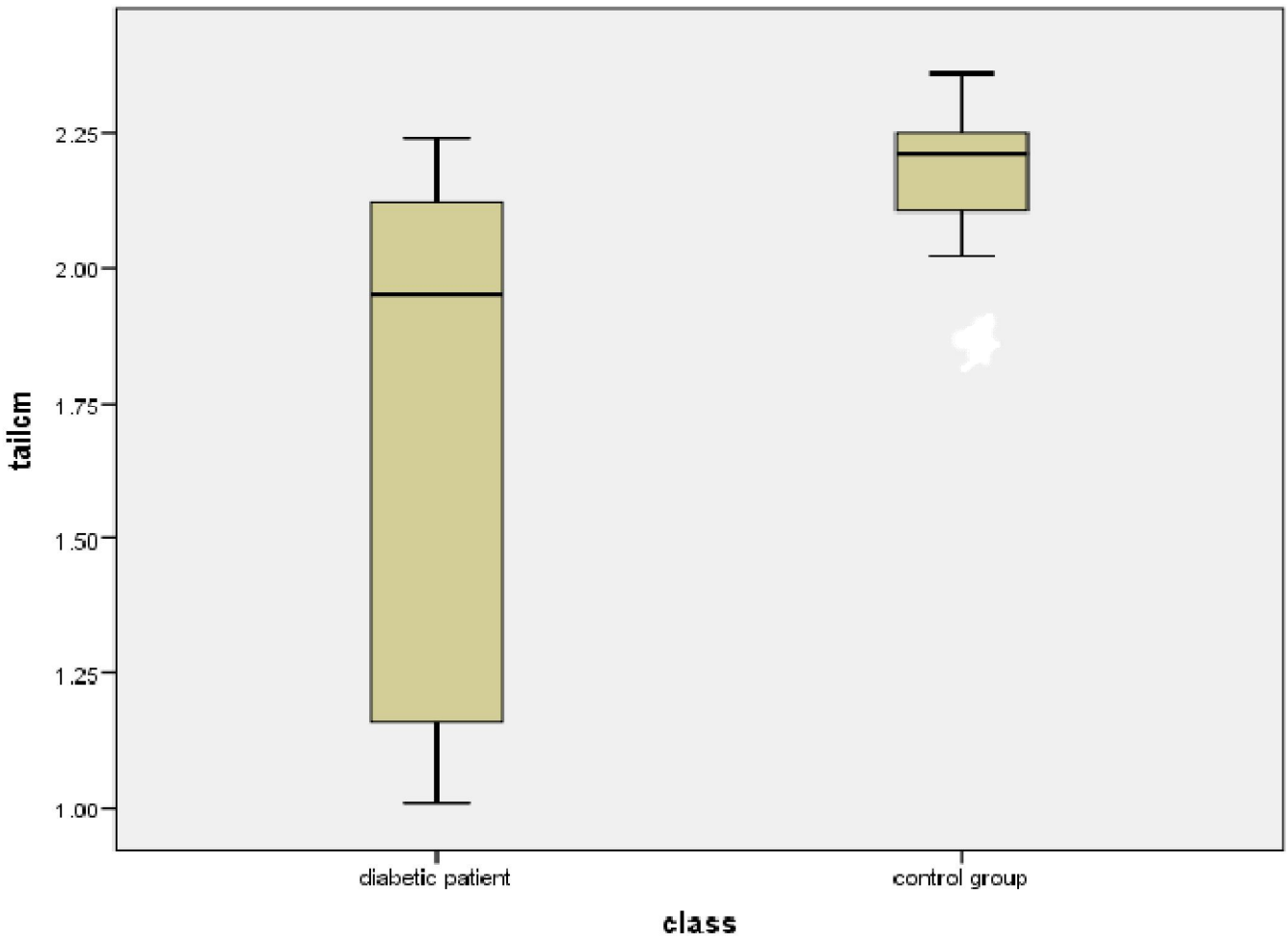
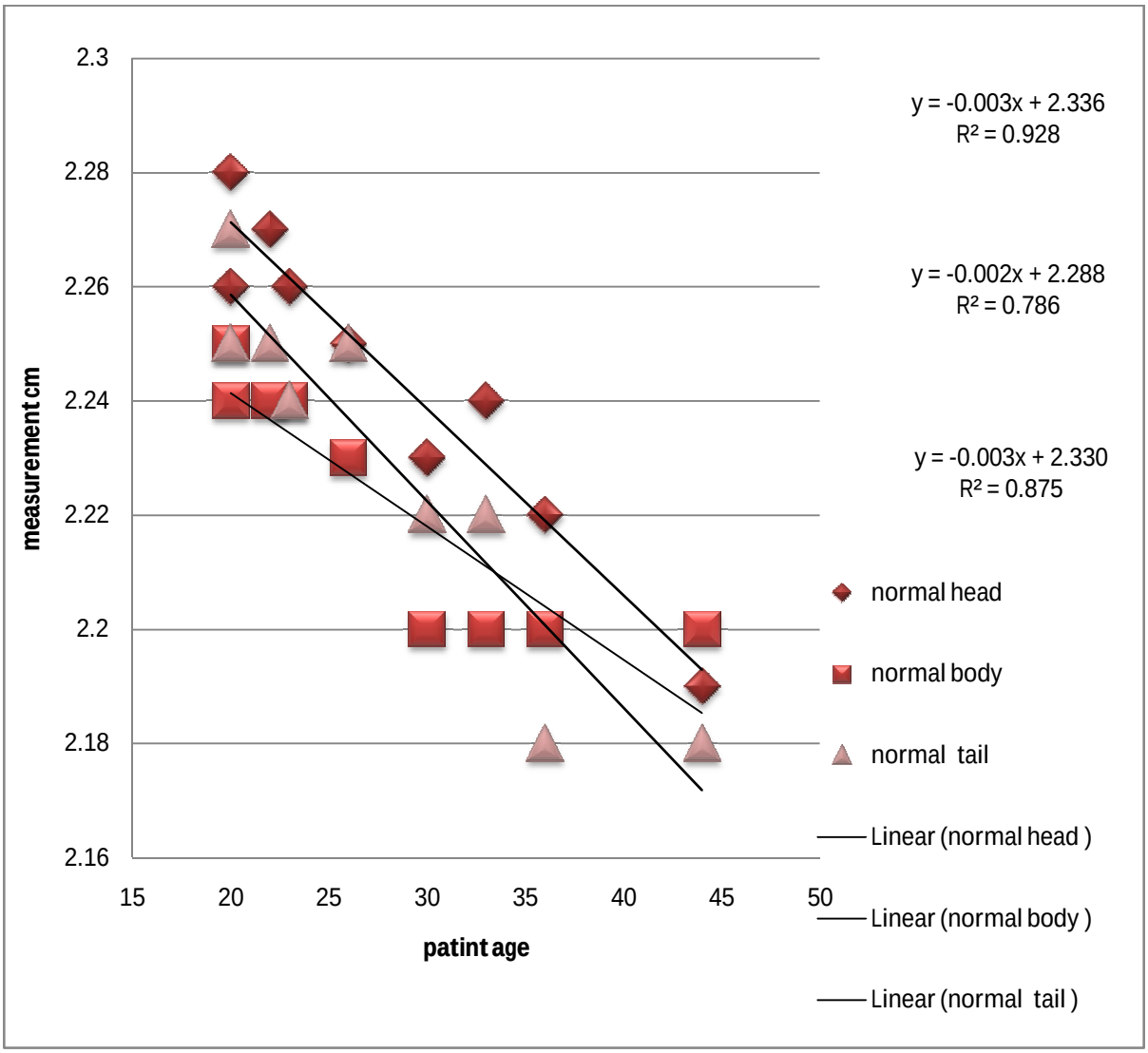
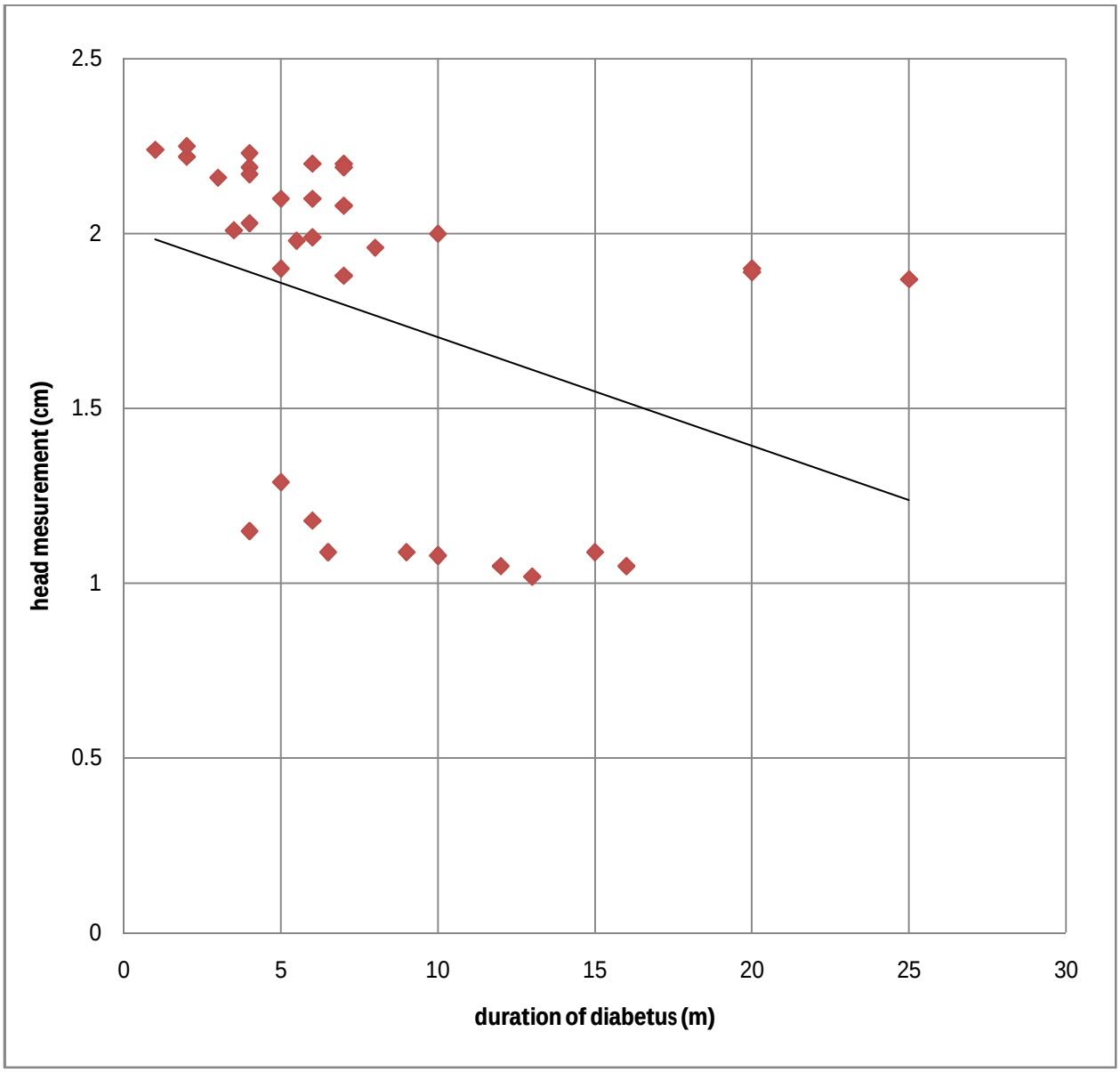


Figure (4.3)box plot shows measure the tail of pancreas in control and diabetic groups.



Figure(4.4) Scatter plot showes the linear relation between the measurement of normal control group and the patient age.



Figure(4.5) Scatter diagram shows the relationship between head measurement (cm) and the duration of diabetes y=-0.0311x+2.0162, R^2 = 0.1349

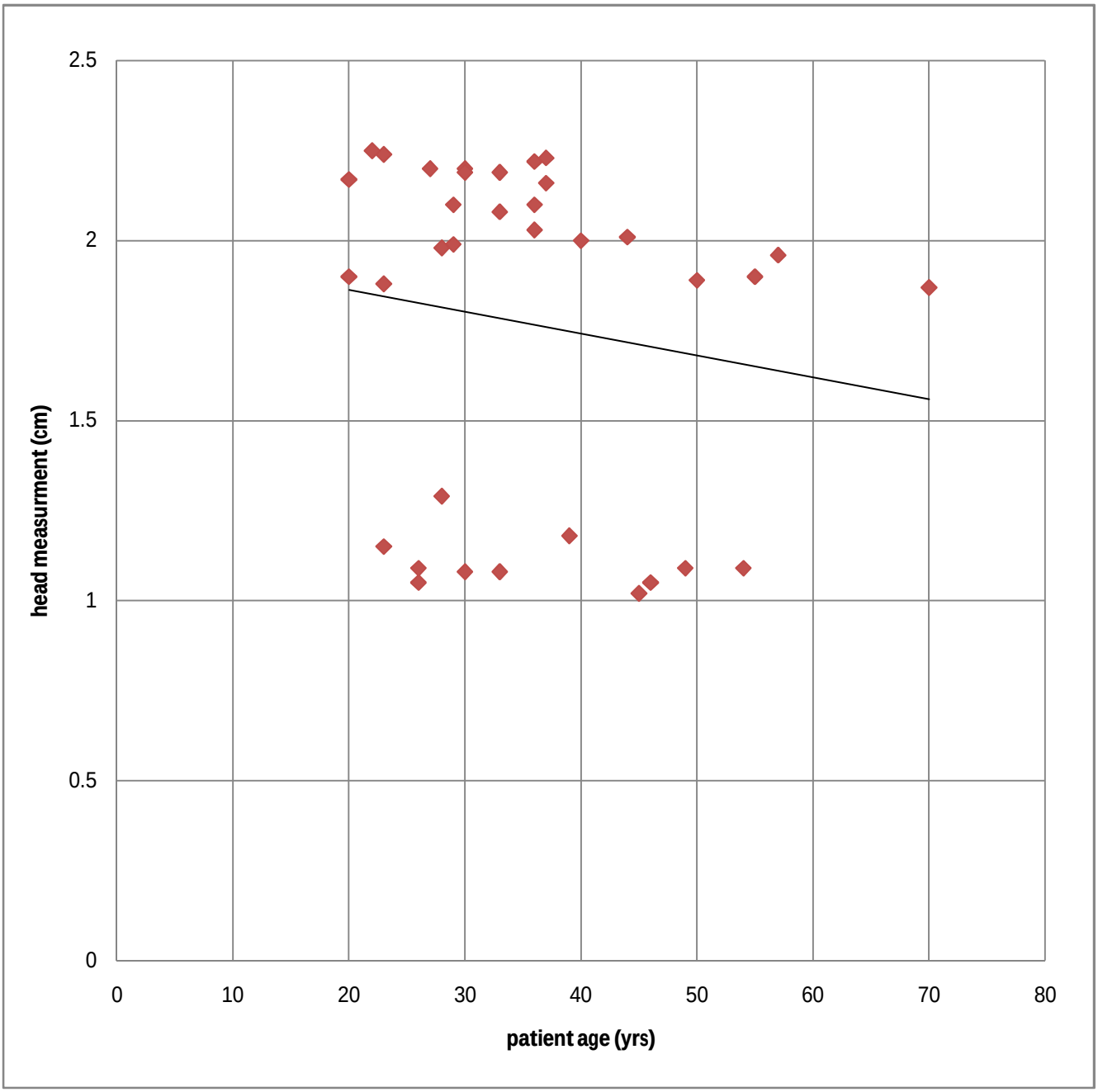


Figure 4.6scatter diagram shows the relationship between head measurement (cm) and age of the patient y=-0.0061x+1.9843, R^2 = 0.0234

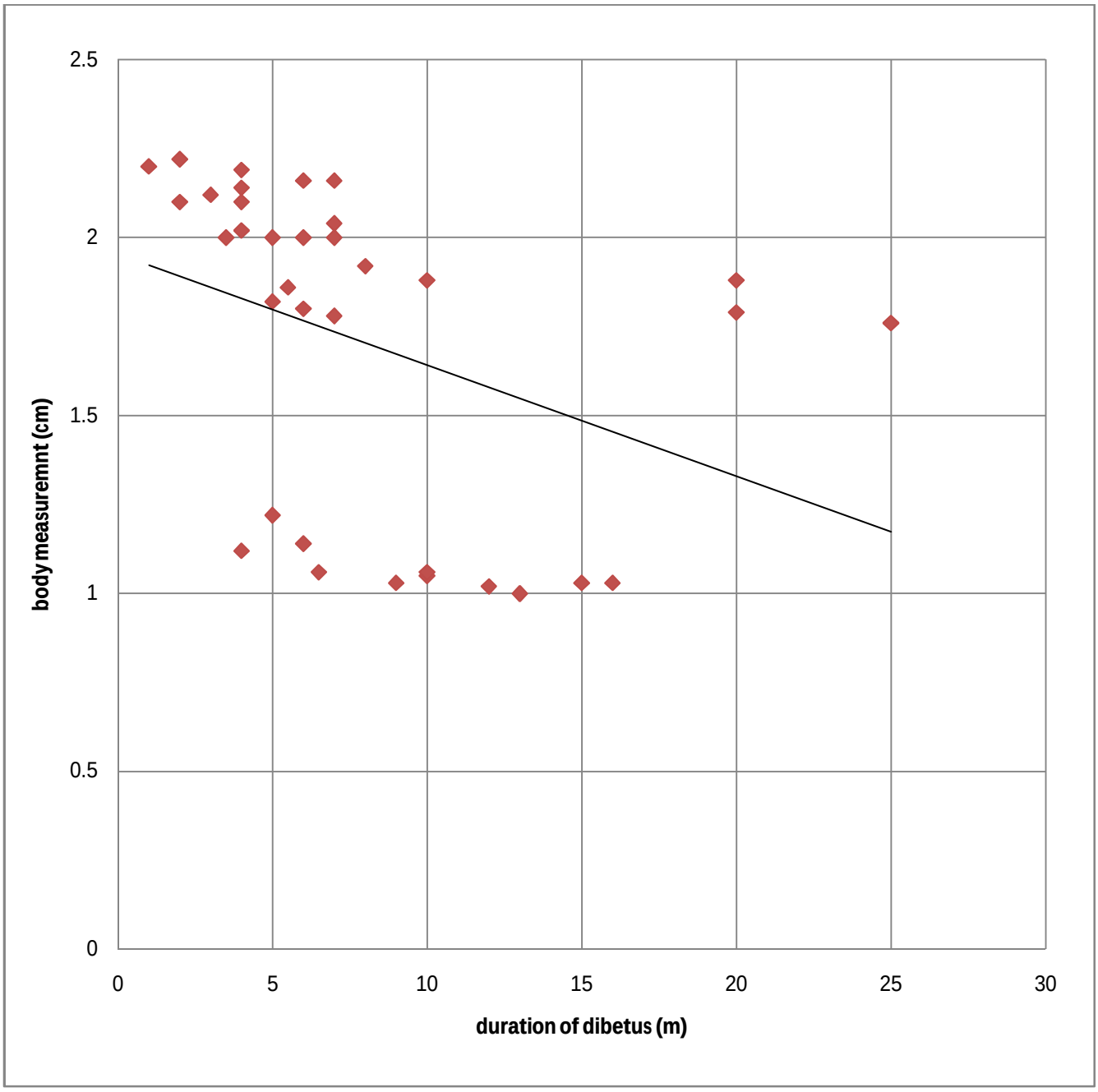


Figure (4.7) scatter diagram shows the relationship between body measurement (cm) and duration of diabetes (month) y=-0.0313x+1.9544, R^2 = 0.1448

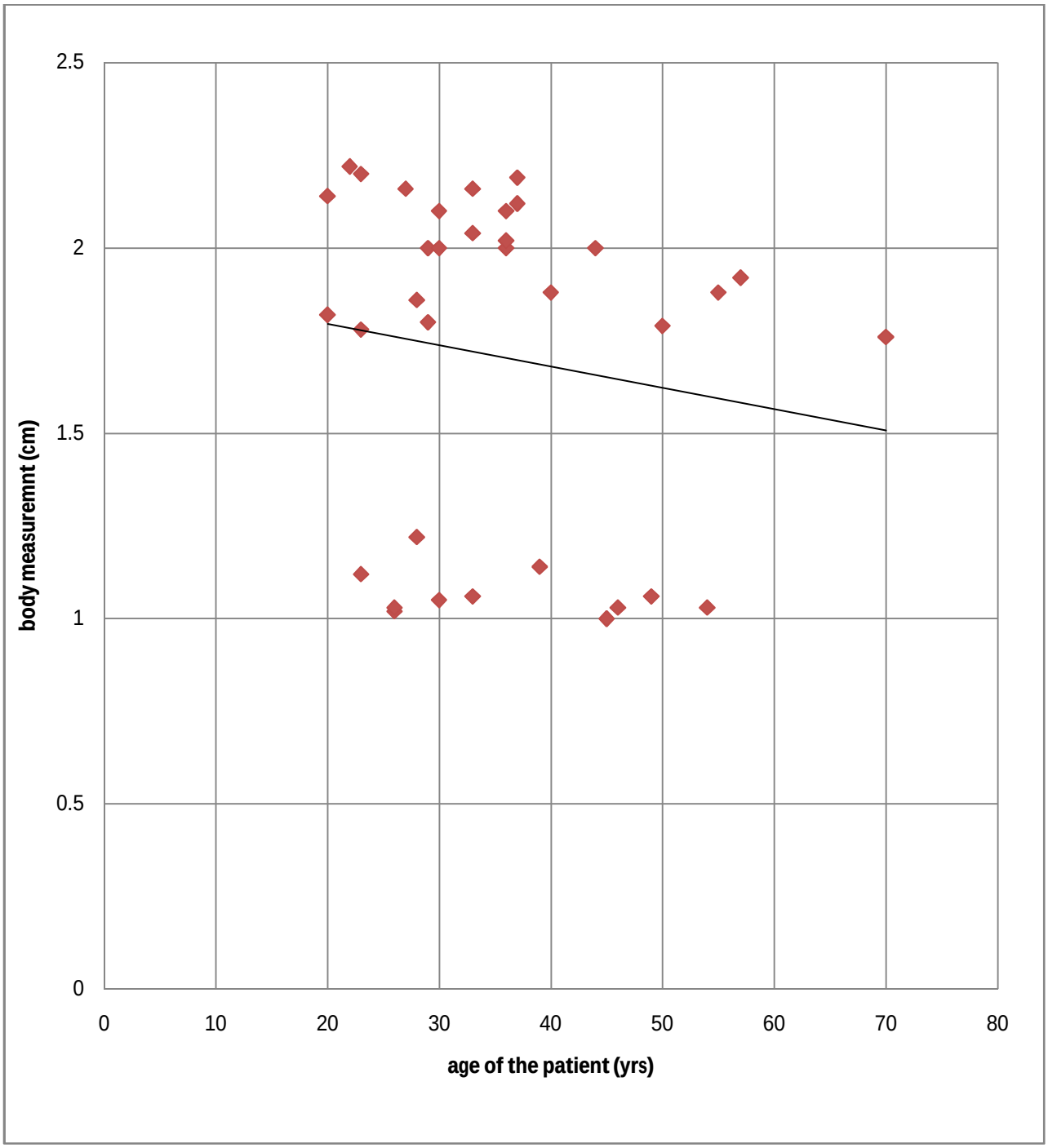


Figure (4.8) Scatter diagram shows the relationship between body measurement (cm) and patient age (yrs.) y=-0.0058x+1.9107, R^2 = 0.0225

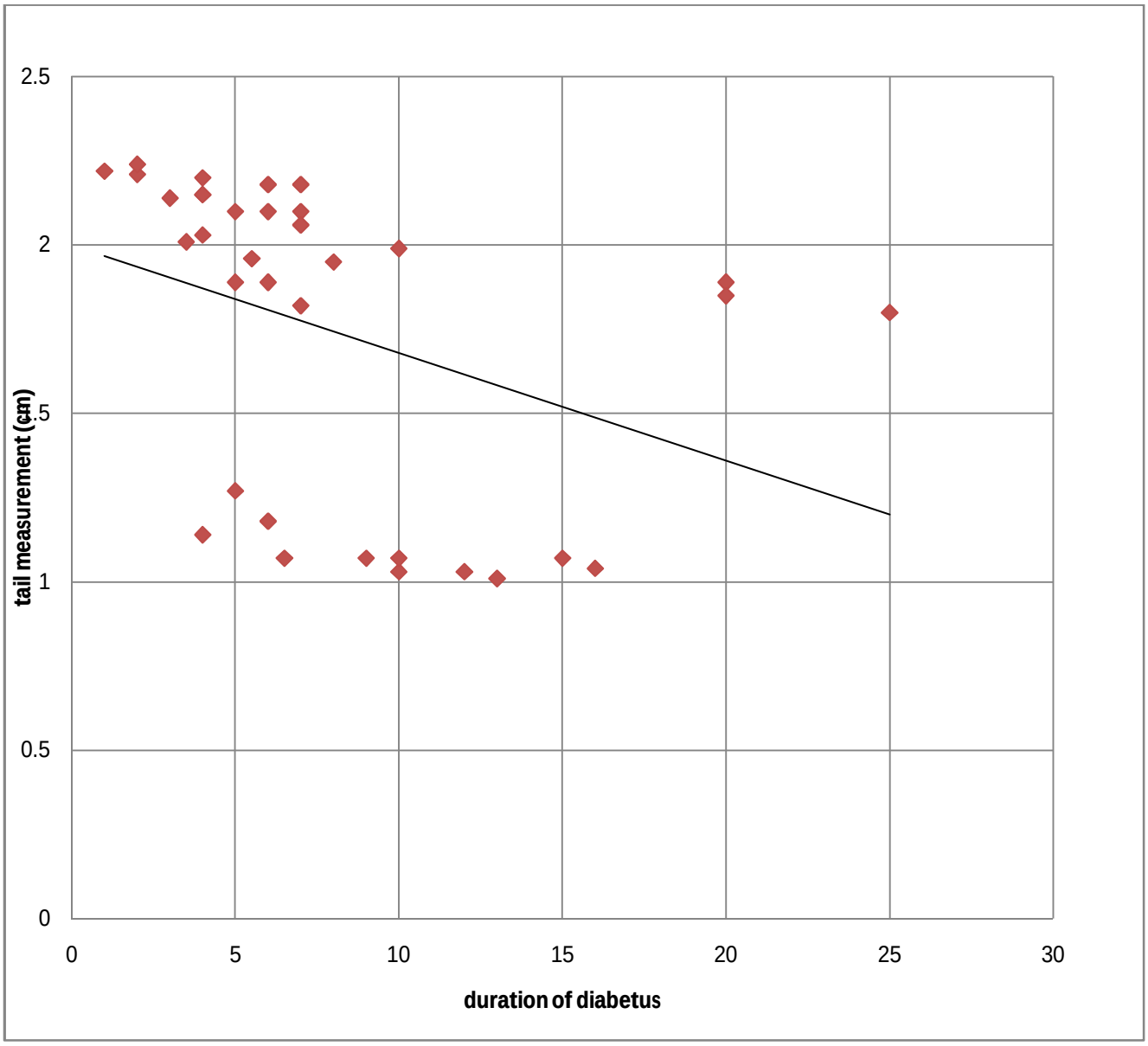
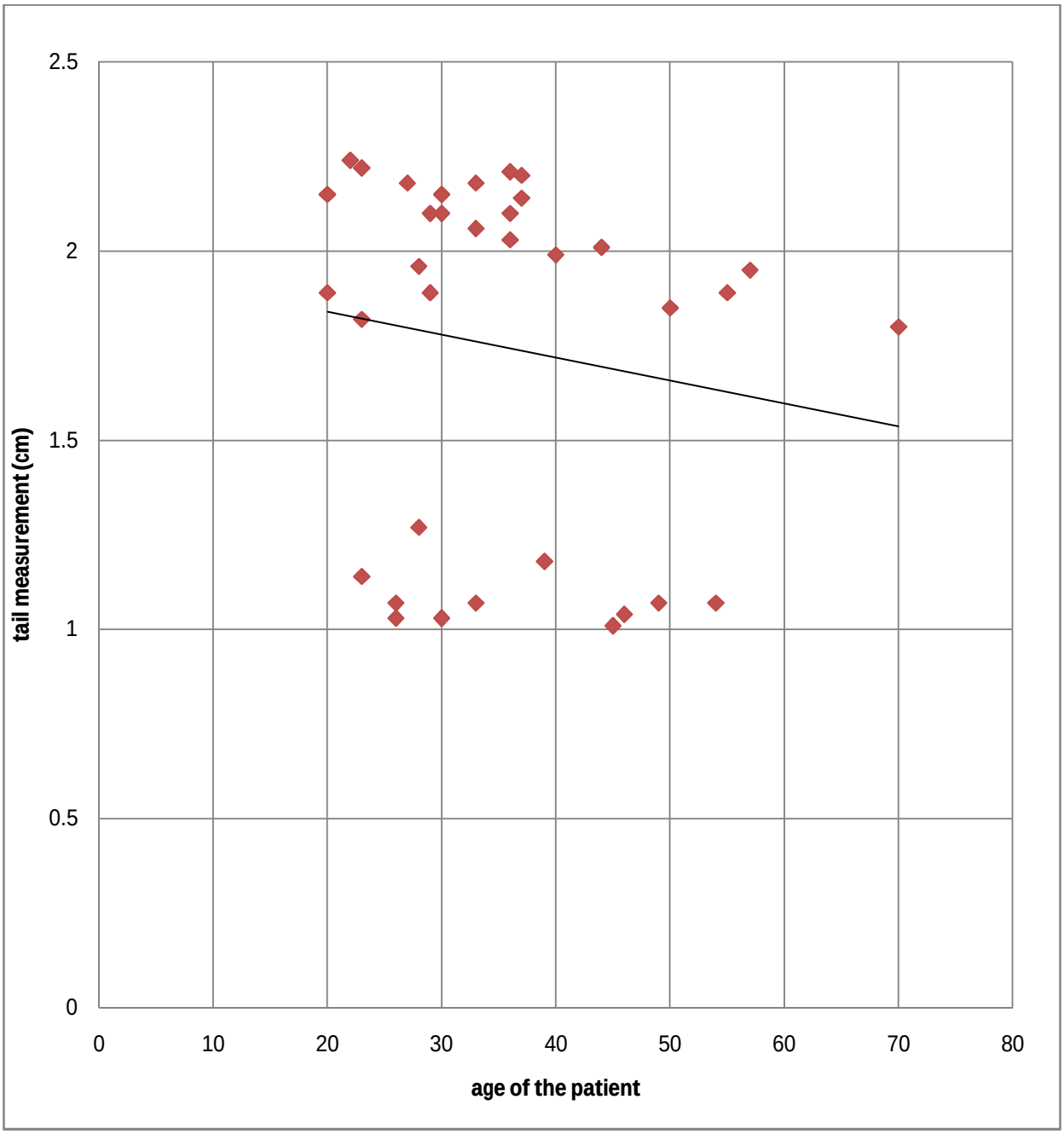


Figure (4.9) Scatter diagram shoes the relationship between tail measurement (cm) and duration of diabetes (month) y=-0.032x+2.0002, R^2 = 0.1447



Figure(4.10) Scatter diagram demonstrate the relationship between body measurement (cm) and duration of diabetes (yrs.) y=-0.0061x+1.9606, R^2 = 0.0236

Chapter five

Discussion, conclusion and recommendations

5.1. Discussion

This study aimed to evaluate the change of pancreatic size in diabetic patients using abdominal ultrasound, more than 70 patient with diabetes underwent U/S examination of the abdomen and the pancreas was evaluated in all patient in sagittal, and transvers section, also same number of the healthy population was examined using the same technique and the head, body and tail was measured using the US machine in order to test the difference between these groups ,the data were collected from Khartoum state Noreen Diabetic center, diagnostic radiology department (U/S section).

The age of control range from (25 to52) and the age of diabetic patient range from (20to 70),and the mean±STD age of these patient was 35.54±11.843 and 29.40±8.605 year for the patient and the healthy people respectively. Also the duration of the diabetes was 7.957±5.5458 years.

Measurement was performed for head, body and tail mean±STD of these was 2.2400±0.02981, 2.1990±0.07325 and 2.2200±0.04422. 1.7689±0.46944, 1.7057±0.45546 and 1.7454±0.46682 for normal and diabetic patient respectively, a significant difference noted between the diabetic patient condition and control group of healthy people and this agree with M Gould et al studywhich stated that significantly different indiabetic patients andcontrol group in pancreatic size measurement.

a linear correlation was done in order to assess these relationshipbetween the pancreatic size and the age of control groupthat result:

There is a strong invers linear relation between the pancreatic size and the age in the normal subject this described as:

The head decreased by -0.0033 (cm) per yearwhile the body decrease by 0.0023cm per year and the tail size decrease by 0.0036cm per year

This also agrees with M Gould et al who stated thatan association between the size of the pancreas and the age, this may be due to normal anatomical variant with age of the people.

Alsomeasurement was correlated with the duration of diabetes in order to investigate the effect of these duration on the size of pancreas, the result showed significant strong inverse relationship between the pancreatic size and the duration of diabetic which the head decreased by 0.0311 cm per year while the body decrease by 0.0311 cm per year ,and the tail decrease by 0.032 cm per and H his study was aimed to evaluate the size of pancreas in diabetes patient a study based on ultrasound ,These results agree with H Green et al stated that the duration of diabetic decrease the size of pancreas.

These study showed no effect of sex on the pancreatic size.

5.2 Conclusion:

This study aimed to evaluate and measure the pancreatic size in diabetic patient and healthy control group, and tested on thirty fife diabetic patients and thirty fife healthy control group, by using ultrasound machine (Mindary-DP2200) with curvilinear 3.5 MHz transducer.

And this study was tested in Noreen Diabetic center (Khartoum, Sudan) from July up to September 2015.

All subjects were scanned for Abdominal Ultrasound in Transvers and longitudinal section.Measurements were obtained at areas including Head, body and tail of pancreas this study include all diabetic patients with diabetic disease only and exclude all diabetic patients with another disease, and analysis the collect data byMicrosoft Excel and SPSS program version.

This study fined there are significant invert relationship between the size of pancreas and the diabetic, the head decease 0.031cm per years ,the body decrease 0.031cm per years and the head decrease 0.032cm per years. and this study fined invert relationship between the size of pancreas anddiabetic duration, and invert relationship between the size of pancreas and the age of patient compare with healthy control group.

5.3. Recommendation:

1. Ultra sound is an operator dependent so that he can need well training to well scanning of pancreas.
2. According to location of pancreas may be obscure by gases, so that the full preparations of patients give good result.
3. The measurements of pancreatic size for diabetic patient must be record and to help the specialist to follow-up the patient treatment plan.

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acinar axis. Diabetes 34:980-86, 2000.

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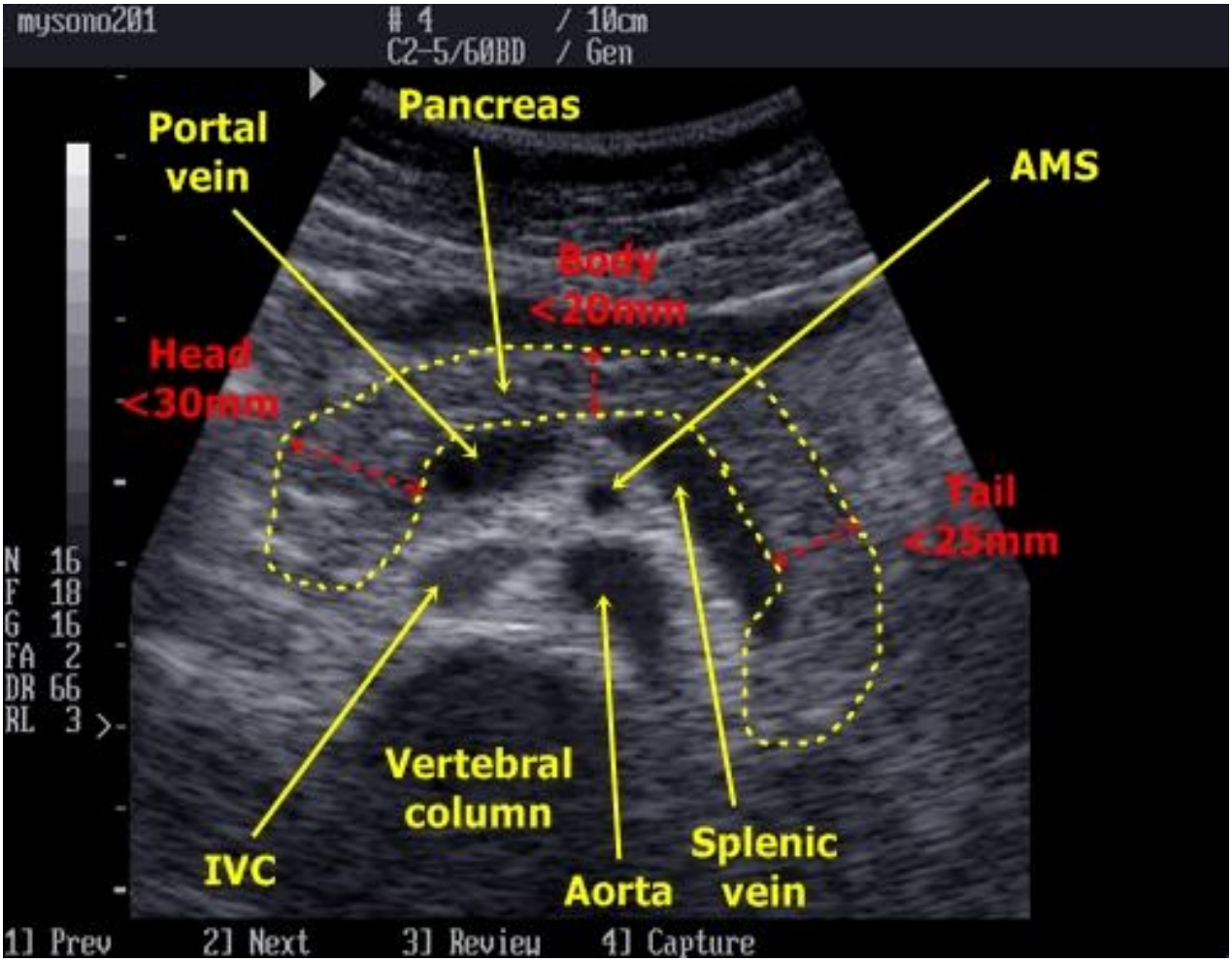
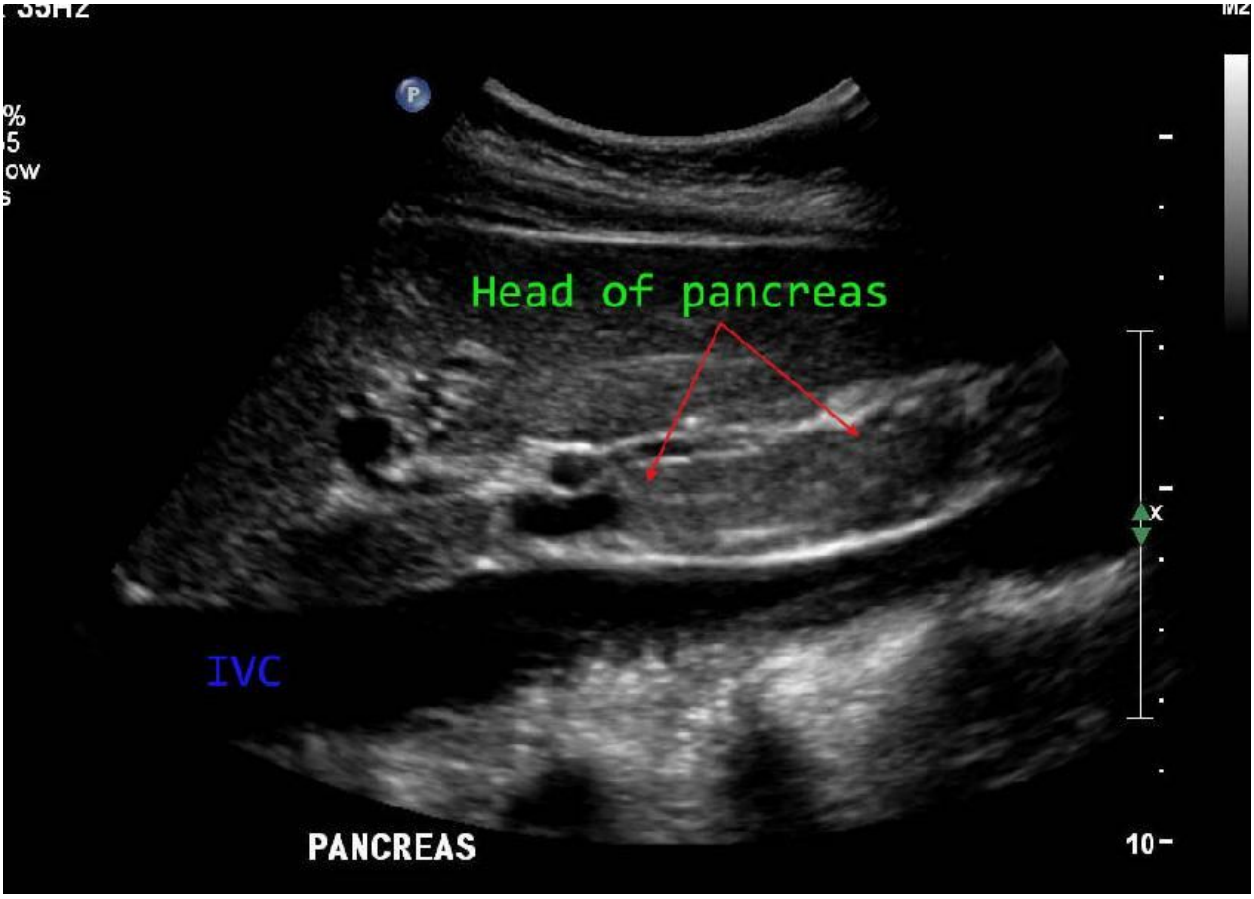
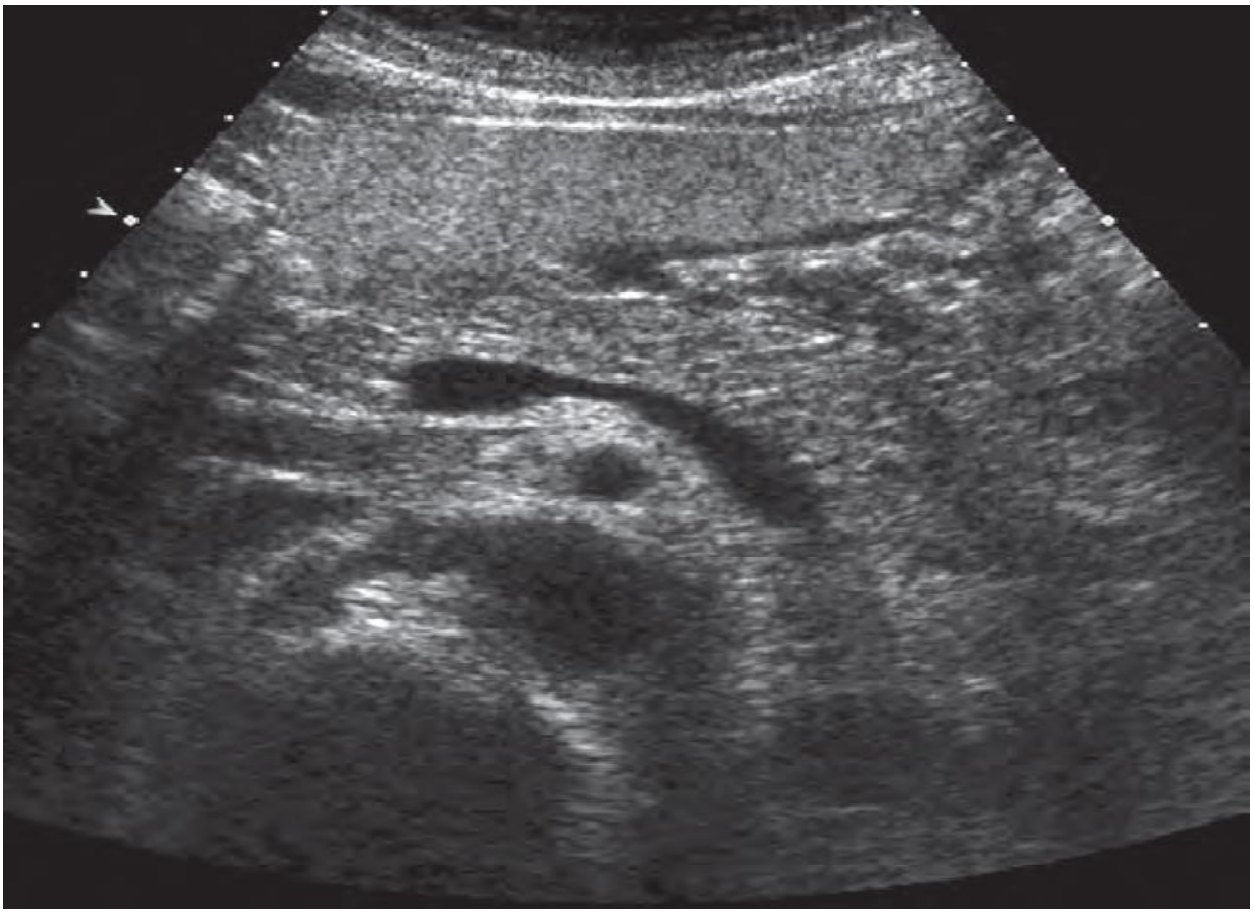
Appendix:



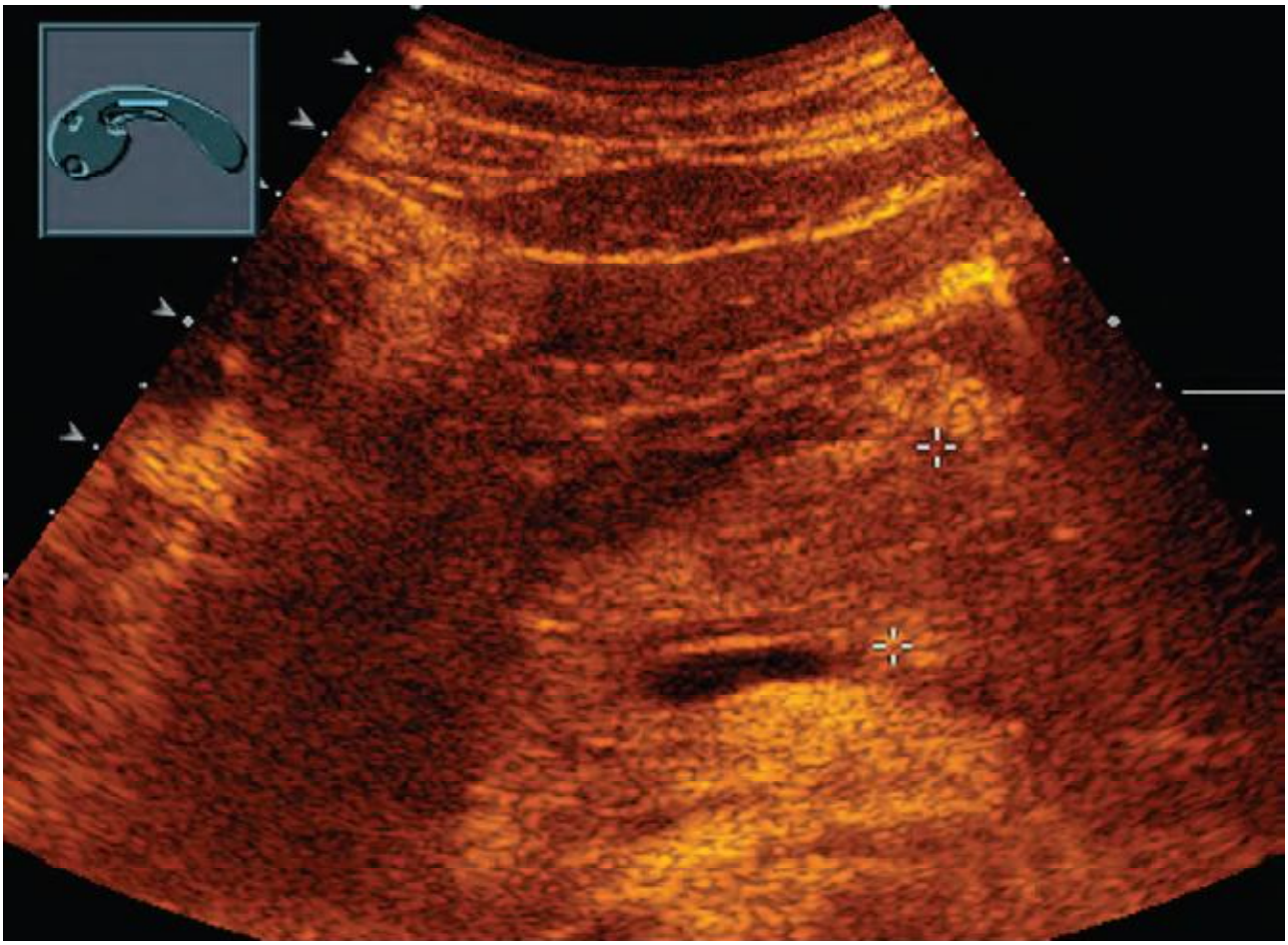
Pancreatic Ultrasound ,transvers section, show normal pancreas components(Head, Body and Tail)



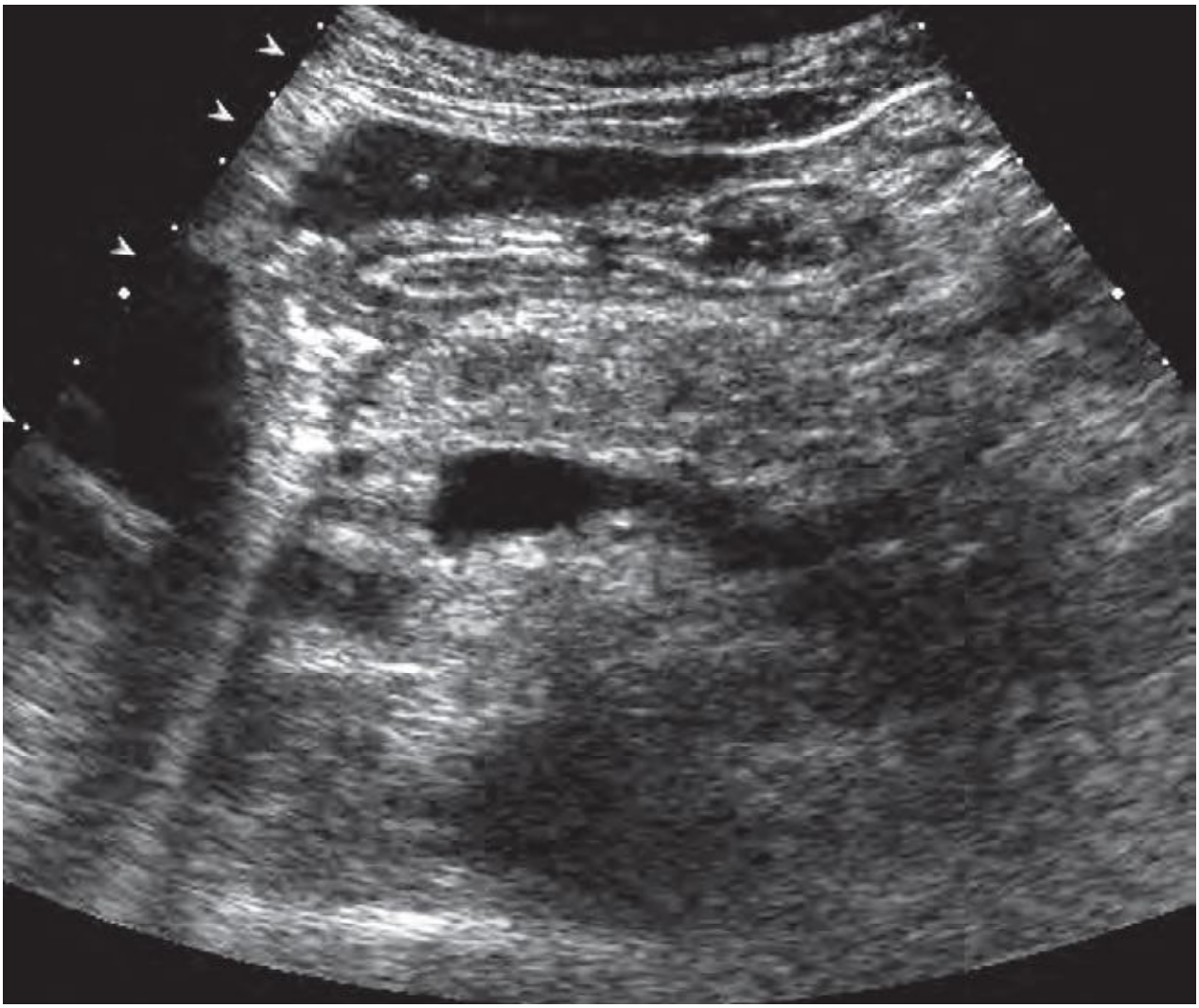
Pancreatic Ultrasound Latitudinal section, show Head of pancreas.



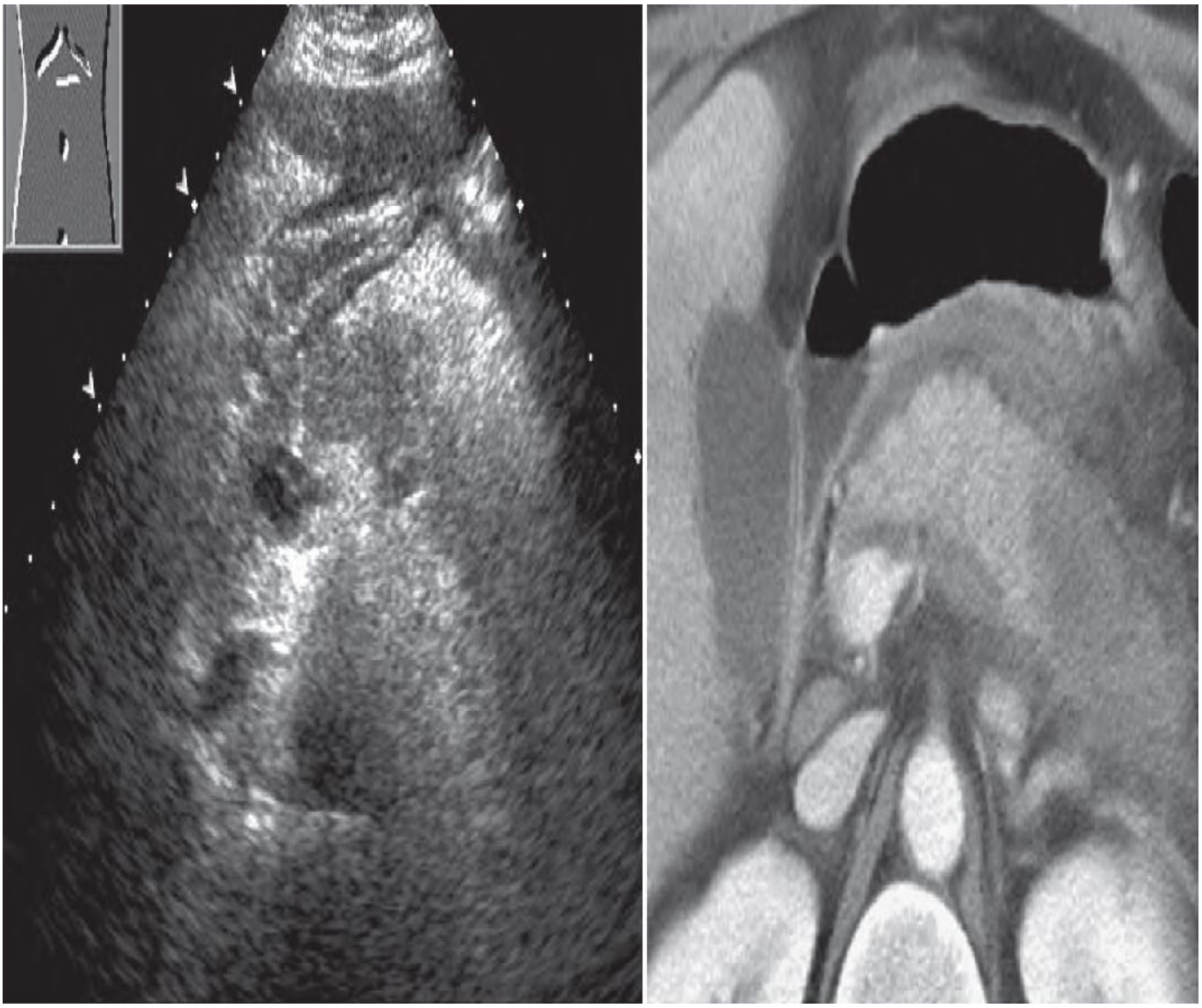
Normal pancreatic body and landmarks.
Transverse image of the pancreatic body and its dorsal landmark, the splenic vein *(white arrow)* and portosplenic confluence. Note the normal parenchymal echogenicity, equal to that of the liver echogenicity. The superior mesenteric artery *(yellowarrow)* is surrounded by a collar of fat. *A,* Aorta; *P,* pancreas



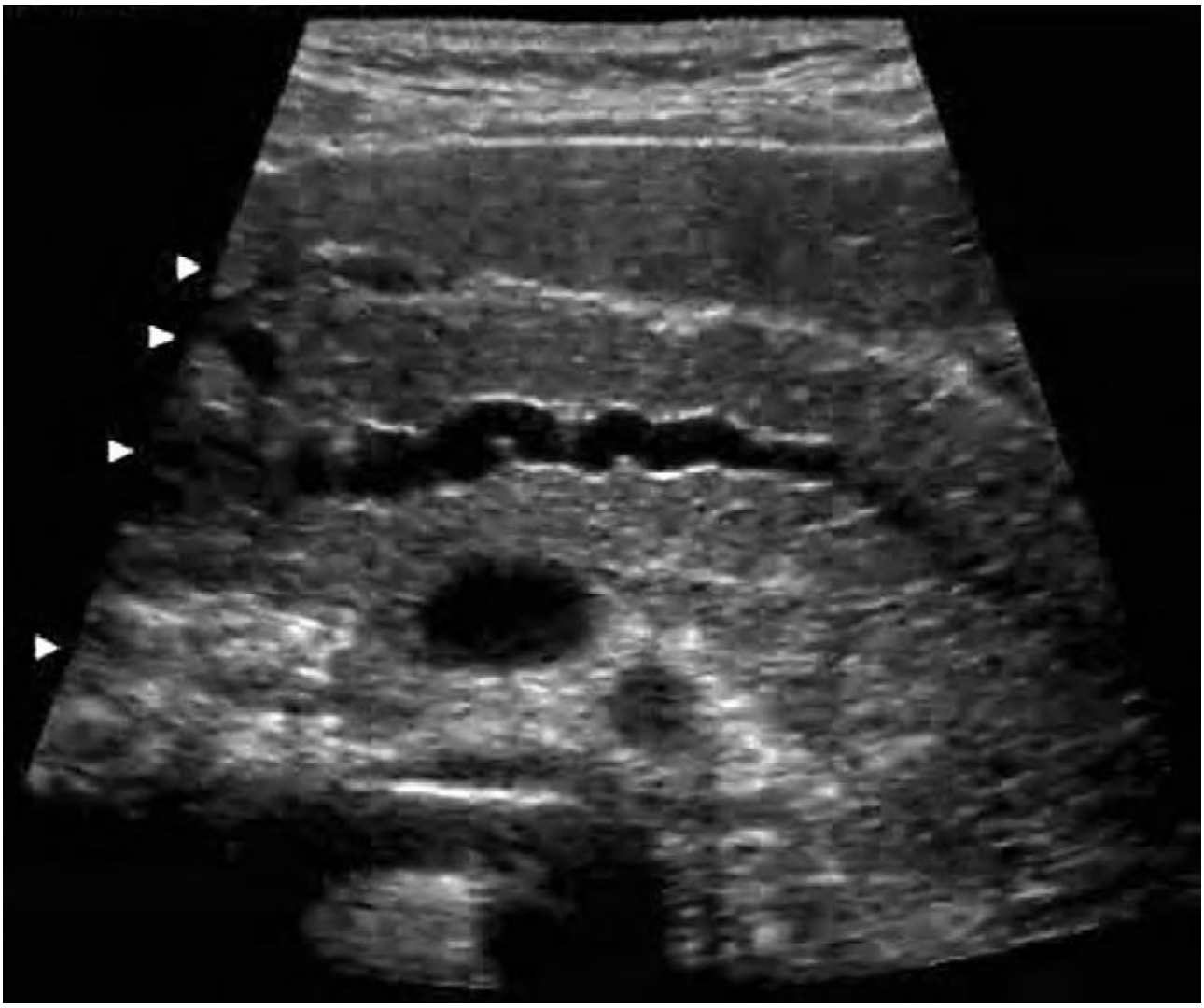
Enlarged pancreas, acute pancreatitis with inflammation. Transverse image of the pancreas shows 26-mm anteroposterior dimension at the level of the superior mesenteric artery *(SMA).* Note the acute inflammation ventral to the pancreas *(yellow arrow)* and ventral to *(blue arrow)* the splenic vein–superior mesenteric vein confluence *(C).*



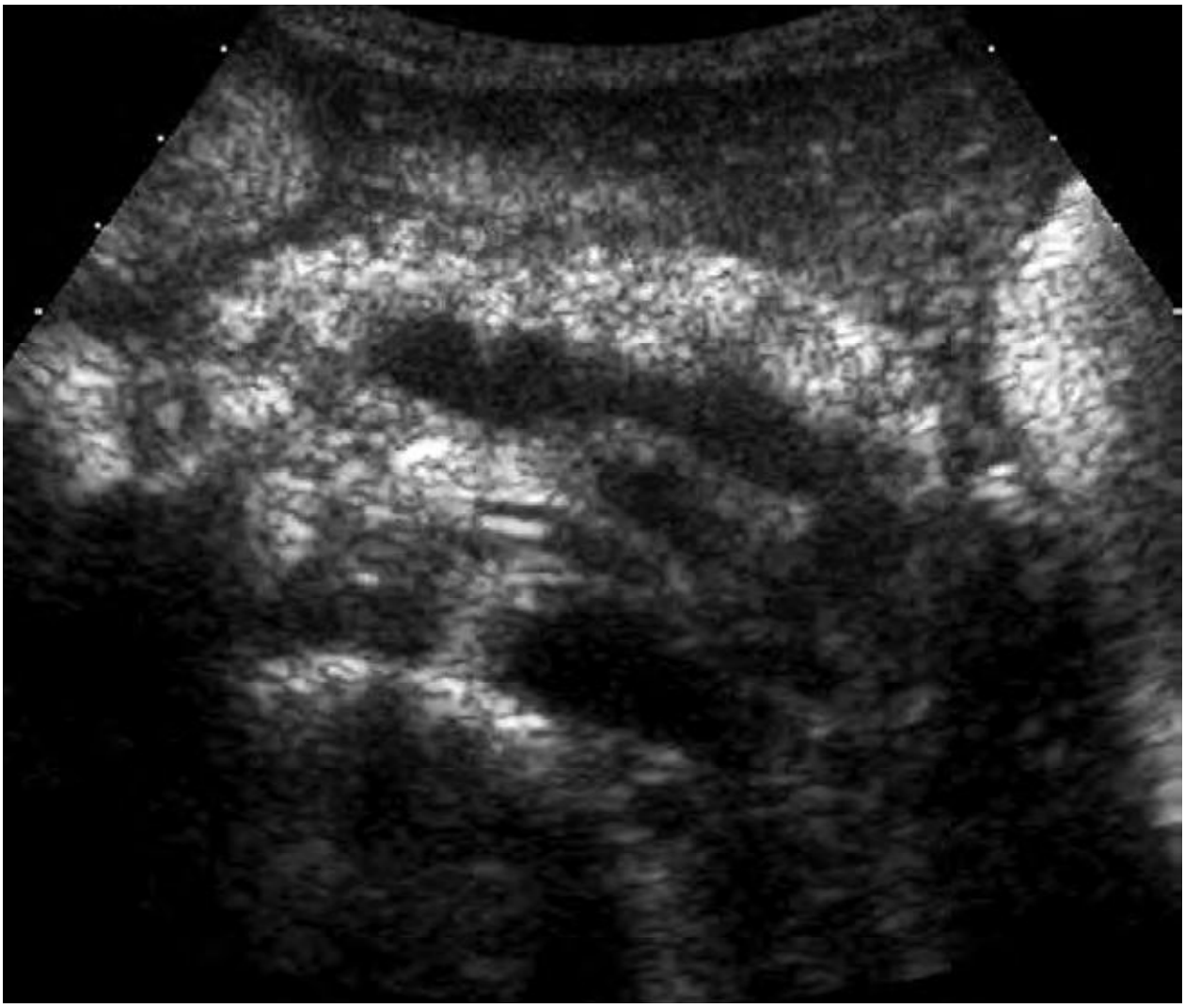
Focal hypoechoic area, acute pancreatitis.
Transverse image shows heterogeneous pancreas with focal hypoechoic area *(arrow).*



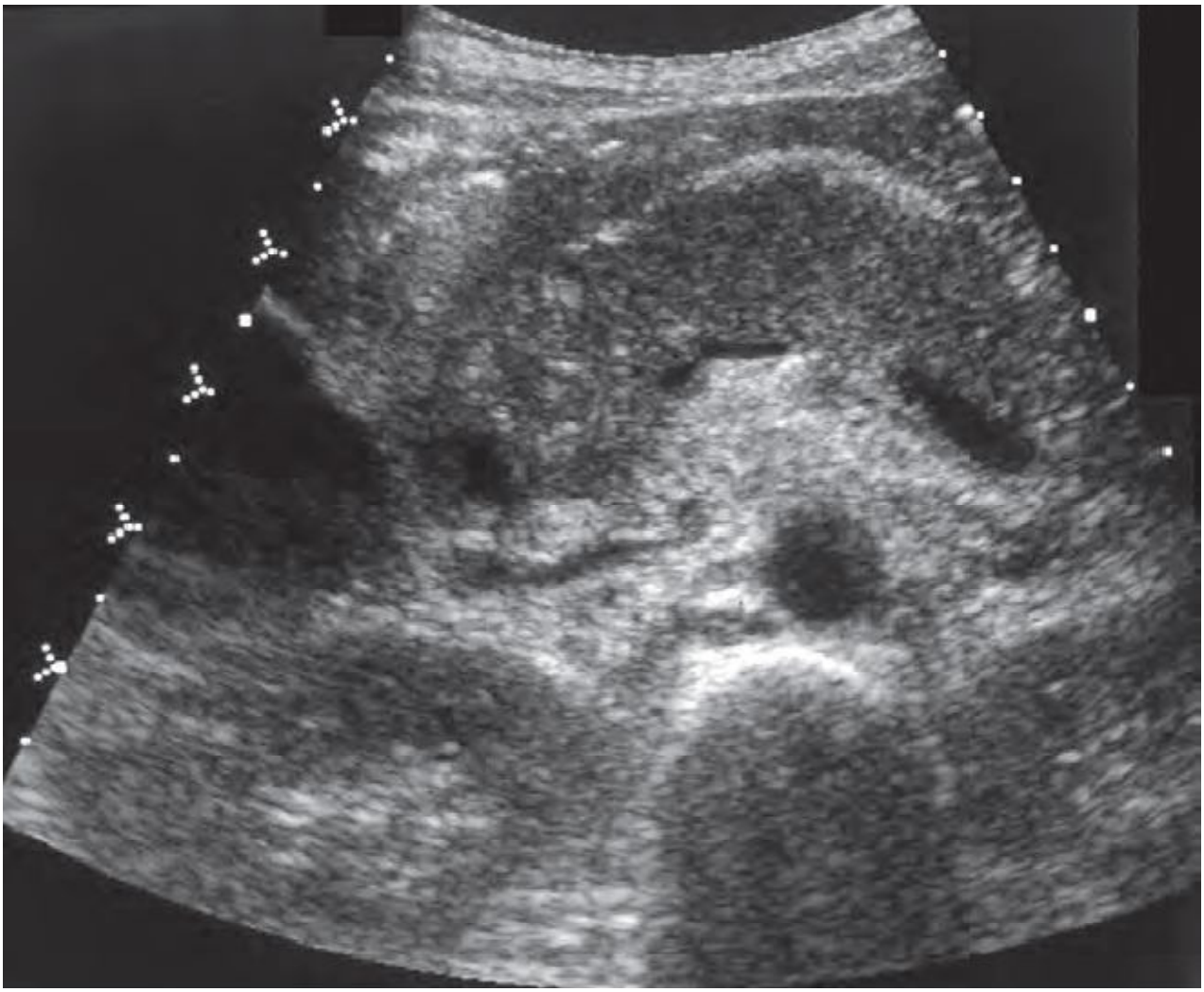
Pancreatitis causes perivascular inflammation. A, Transverse sonogram, and **B,** CT image, of the pancreasshow obvious evidence of perivascular inflammation *(arrows)*, hypoechoic on sonogram and low density on CT.



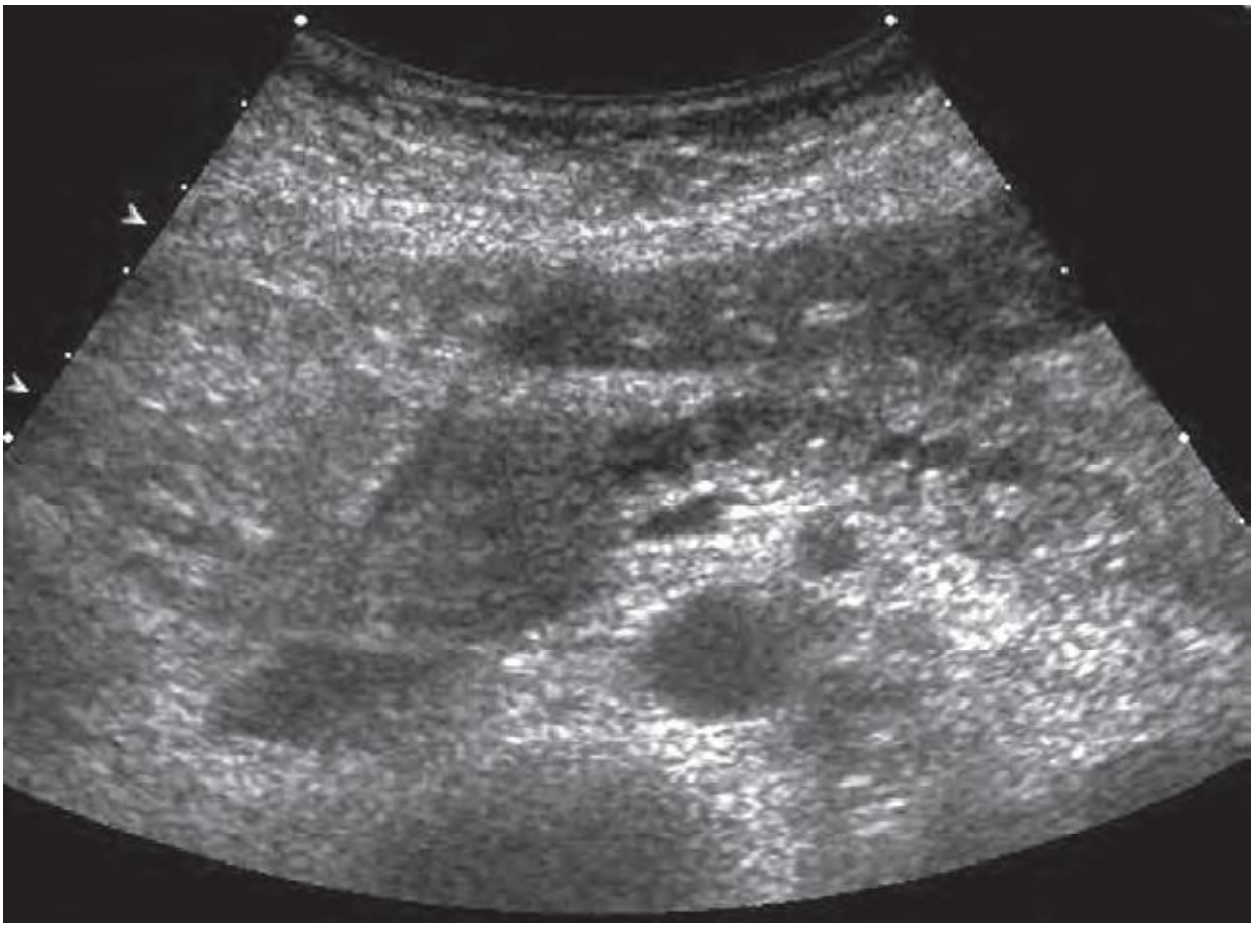
Dilation of pancreatic duct. Transverse sonogram of the pancreatic body shows a beaded, dilated pancreatic duct, resulting from chronic pancreatitis.



Dilated pancreatic duct and branch duct calcifications.
Ductal calcification is a hallmark ofchronic pancreatitis. Transverse sonogram shows many branchduct stones.



Diffuse pancreatic carcinoma.
Transverse sonogram; fewer than 5% of all ductal adenocarcinomas are diffuse.



Pancreatic carcinoma. Transverse sonogram shows hypoechoic mass obstructing the pancreatic duct. Note the “beaded” pattern of the dilated, obstructed duct. *A,*Aorta; *IVC,* inferior vena cava.