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Nov. 15, 1930.] THE BRITISH MEDICAL JOURNAL. 35

VIEW OF THE INTERIOR OF THE MOSQUE OF CORDOVA.—Buildings raised by or for the Arabians in the countries which they occupied were often modelled upon the local type, but modified to suit their needs or tastes. This resulted in originality. Moreover, the Arabians often took their materials from ancient structures on the spot, and the adaption of these alone led to important developments. Thus, columns found to be too short for the purpose were prolonged by placing vertical pieces between the capital and the spring of the arch, or even by superimposing a second column upon the capital; they masked these devices by making them the basis for skilful combinations of arcades.

DATE: A.D. c. 960

Carbimazole 2008-2

Question 5: Thioamides	1. How does carbimazole act in thyroid disease?	Metabolised to methimazole: **Major action block hormone synthesis** T3 and T4 **Inhibits thyroid peroxidase** – limits organification of iodine. Also blocks coupling of iodotyrosines Small action in blocking peripheral deiodination of T3 and T4. Slow onset as T4 may takes weeks to become depleted	Bold to pass
	2. What are the major side effects of carbimazole?	Rash maculopapular, pruritus – common; **B one marrow suppression**: neutropenia, agranulocytosis (reversible). Others – urticaria, arthralgia, lupus reaction, vasculitis, jaundice/hepatitis; nausea and GI, occur early	1 side effect
	3. How does carbimazole differ from propylthiouracil?	**Carbimazole is a prodrug** - converted to methimazole in vivo. Methimazole is 10 times more potent **And one of the areas below** 1. PTU has greater action in inhibiting peripheral deiodination of T4 and T3 2. Propylthiouracil is strongly protein bound: preferred in pregnancy; not secreted in breast milk 3. PTU has shorter half life 1.5 vs 6 hours. PTU given qid, Carbimazole is daily 4. PTU bioavail 50-80%, vs Carb 100% Vd = TBW) 5. PTU excreted in urine as glucuronide metabolite <24 hours, carb in 48+ hours)	Bonus marks

Corticosteriods 2014-1-B

Stem: Moving now to your pharmacology question. Your planned treatment includes IV hydrocortisone.			
Question 3 Corticosteroids **Subject:** Pharm LOA: 1	Describe the mechanism of action of corticosteroids at a cellular level?	• Most of known effects via widely distributed **glucocorticoid receptors** • Present in blood in bound form on Corticosteroid Binding Globulin (CBG) • Enters cell as free molecule • Intracellular receptor bound to stabilizing proteins (most important heat shock protein 90, Hsp90) • Complex binds molecule of cortisol then actively transported into nucleus where binds to **Glucocorticoid Receptor Elements (GRE)** on the gene • Interacts with DNA and nuclear proteins regulating transcription. Resulting mRNA exported to cytoplasm for **protein production** for final hormone response	Bold to pass
	How can corticosteroids be classified? Prompt: How do they differ in their action?	1. length of action (hydrocortisone short to medium-acting, dexamethasone or betamethasone long-acting) 2. **anti-inflammatory activity** (potency: hydrocortisone 1, prednisolone 5, dexamethasone 30) 3. **mineralocorticoid activity** ie., salt retaining (fludrocortisones 250 times that of hydrocortisone) 4. topical vs non topical	bold
	What are the side effects of corticosteroid use? Prompt: what about long term effects?	- Short term: (<2 weeks): insomnia, behaviour changes, acute peptic ulcer, acute pancreatitis, hyperglycaemia - Long term: - Cushing's Syndrome (moon facies, fat redistribution, fine hair growth, acne) secondary to hormonal actions. (Rate of development function of dose and genetic background) - hyperglycaemia, diabetes - myopathy - osteoporosis, aseptic necrosis - psychiatric (hypomania, acute psychosis, depression) - Na,fluid retention, K+ loss - **adrenal suppression** / addisonian crisis - poor wound healing **- immunosuppressant**	Bold and 4 others

Dexamethasone 2016-2-D

Stem: He is treated with Dexamethasone. Moving on to Pharmacology.			
Question 5 Dexamethasone (Dose/PD) **Subject:** Pharm LOA: 1	a. What is the usual dose of dexamethasone for treatment of croup?	0.15-0.60mg/kg PO, single dose	dose range to pass
	b. How can dexamethasone be administered? *Prompt: Any other routes?*	Oral, IV, IM, topical	3/4 to pass
	c. How does the anti-inflammatory effect of dexamethasone compare to hydrocortisone? *Prompt: How does the duration of action differ?*	Dexamethasone is 30 times more **potent** and **longer acting**	Bold to pass
	d. Describe the anti-inflammatory and immunosuppressive effects of glucocorticoids.	Effects on concentration, distribution and function of peripheral leucocytes Suppression of inflammatory mediators (cytokines, chemokines) Inhibit function of macrophages and antigen presenting cells Inhibit PLA2 -> decrease PG/LT/PAF	2/4 to pass

Dexamethasone 2006-2

3. Dexamethasone	**1.What are the pharmacological differences between dexamethasone and hydrocortisone?**	**1. 30x greater anti-inflammatory potency** **2. Longer duration of action** **3. No salt retaining activity**	
	2.In what situations could you use dexamethasone (3 EXAMPLES FOR PASS)	**1. Diagnosis – dexa suppression test** **2. Anti inflammatory effect (see table 39-2 p651)** **3. Croup**	

Diabetes Mellitus Drugs 2009-2

Question 2: **Classification of drugs used in diabetes mellitus**	(a) Outline the groups of drugs that are used to treat hyperglycaemia in diabetes mellitus.	• **Insulin** • **Sulfonylureas -** • **Biguanides** • Meglitinides • D- phenylalanine derivatives • Thiazolidinediones • Alpha-glucosidase inhibitors	Must get 3 bolded groups to pass.
	(b) Contrast the mechanism of action of sulfonylureas and biguanides.	Sulfonylurea: • **Increase insulin release from pancreas** • Reduction of serum glucagon levels • Closure of potassium channels in extrapancreatic tissues Biguanide: • **Action does not depend on functioning pancreatic B cells** • May directly stimulate glycolysis in tissues with increased glucose removal from blood; • May reduce hepatic gluconeogenesis; • May slow of absorption of glucose from the GI tract; • May reduce glucagon levels	Bold to pass

TOPIC: Glucagon & its role as an antidote NUMBER: 5

OPENING QUESTION	**Describe the pharmacologic effects of glucagon**	**Comment**
POINTS REQUIRED	1. Metabolic – • Binds with receptors on liver cells (G protein-linked ↑ adenylyl cyclase & cAMP) • Catabolism of stored glycogen, raising blood glucose level • No effect on skeletal muscle • Also cause release of insulin from B-cells, catecholamines from Pheo and calcitonin from medullary carcinoma cells 2. Cardiac effects • Potent inotropic & chronotropic effect on heart via cAMP without requiring functioning beta-receptor 3. Large doses of glucagon produce relaxation of smooth muscle (not via cAMP)	2 of 3 to pass
SECOND QUESTION	**What are the indications for using glucagon clinically?**	
POINTS REQUIRED	1. Severe hypoglycaemia 2. Overdose on Betablockers (5-10mg IV will reverse hypotension/bradycardia) 3. Relaxation of intestine during radiology of bowel 4. Diagnosing endocrine disorder- • Type I Diabetes (no C-peptide response to glucagen) • Suspected tumours, eg Insulinoma, Pheo, medullary carcinoma (cause rise in hormone)	2 out of 3 to pass
ADDITIONAL QUESTIONS	**What are the adverse reactions produced by glucagon?**	
	• Transient nausea & vomiting • Relatively free from severe adverse reaction • hyperglycaemia	

Glucagon 2003-2

Glucagon pp730-2	Regarding glucagon outline its pharmacodynamic effects and relate these to its clinical use.	• Glycogenolysis and gluconeogenesis thus increasing serum glucose –treatment of hypoglycaemia • Positive ionotropic and chronotropic effect on the heart via glucagon receptors and cAMP – treatment of B Blocker OD. • Relaxation of intestinal smooth muscle – treatment of food bolus obstruction or to aid radiology of the bowel	To pass: must get hypoglycaemia and one other, others bonus	

Hydrocortisone 2011-2

Question 5 Adrenocorticoids (Hydrocortisone)	a) What are the effects of hydrocortisone? (Prompt: *Describe the anti-inflammatory and immunosuppressant effects of hydrocortisone)* b) What are the effects of chronic steroid use?	a) Mediated by glucocorticoid receptors Physiologic + permissive effects Metabolic effects Catabolic and anti-anabolic effects Anti-inflammatory + immunosuppressive effects Other effects: CNS, pituitary axis, psychiatric, renal, neonatal lung Effect concentration, distribution + **function of peripheral leukocytes** **Suppress inflammatory mediators** (cytokines + chemokines, as well as PGs + leukotrienes) Inhibit tissue macrophages + APCs Suppress mast cell degranulation Reduce antibody production (in large doses) **c) Cushings Syndrome** Metabolic effects (moon face, fat redistribution, striae, weight gain, myopathy, muscle wasting, thin skin, bruising, hyperglycaemia, osteoporosis, diabetes, aseptic necrosis, wound healing impaired Other effects (peptic ulcers, psychosis, depression, cataracts, glaucoma, salt retention, hypertension) **Adrenal suppression** (> 2 weeks dosage)	Bolded + one other Bolded + 3 others

Hydrocortisone 2003-2

5. Hydro-cortisone	1. Regarding hydrocortisone, what are its pharmacodynamics? Describe the anti-inflammatory and immunosuppressant effects of hydrocortisone?	• Anti-inflammatory • Immunosuppressive • Catabolic effects • Permissive effects • Metabolic effects • Other: endo, psych Altered leucocyte concentration, distribution and function Inhibit macrophages and antigen presenting cells Reduce interleukins and other mediators Phospholipase A2 and COX 2 Decrease histamine release by mast cells, etc Reduce Ab production	Pass = First 2 plus 1. Pass = two	
	3.What are the effects of chronic steroid use?	Cushings	Several features	

Insulin 2017-1-C

Stem: She is a Type 1 diabetic. Moving on to Pharmacology			
Question 2 Insulin **Subject:** Pharm LOA: 1	a) What is the mechanism of action of insulin? b) What different formulations of insulins are there (prompt - please describe in terms of duration of action and name an example from each group) c) How are their differing properties used to optimize glycaemic control d) Give another Emergency Department use for insulin other than the regulation of blood glucose	a) **Promotes the uptake of glucose from blood into tissues,** esp. fat, liver, and skeletal muscle and promotes glycogen synthesis (Insulin receptors found on cell membranes) **b) Rapid and short acting.** Clear solution, neutral pH, contain Zn, rapid onset, short duration of action. examples: insulin neutral, insulin lispro, insulin glulisine **Intermediate acting** Turbid solution, neutral pH, protamine in phosphate buffer (NPH) to prolong action examples: insulin isophane, insulin aspart protamine **Long acting** Clear solution, soluble, slow onset, prolonged action. Daily administration mimics basal insulin secretion examples: insulin glargine, insulin detemir **c) Combination of insulins with different durations of actions** aim to replace basal insulin requirement (50%) and meal requirement (50%). d) Management of hyperkalaemia, Ca^{++} channel blocker overdose (+/- Beta-blocker)	Bold to pass Bold to pass plus 2 of 3 correct insulins Concept to pass One alternate use to pass

Insulin 2014-1-C

Stem: We now move onto pharmacology.			
Question 2 Insulins (Katzung 12th ed pp 747-753) **Subject:** Pharm LOA: 1	What pharmacological methods are used to optimise blood sugar control when administering insulin? Prompt: what are the different types of insulin?	1. **Titration of dose to BSL** 2. **Pharmacological manipulation of human insulin molecule**: rapid-acting (aa reversal/substitution reducing aggregation properties), intermediate acting (insulin/protamine complexes), long acting (aa substitutions, molecular attachments) 3. Mixing of insulin preparations 4. Continuous subcutaneous insulin infusion devices	Bold to pass
	What are the complications of insulin administration?	**Hypoglycaemia** Hypoglycaemic unawareness Insulin allergy (usually due to non-insulin contaminants) Immune insulin resistance Lipodystrophy at injection sites	Bold + 1 to pass

Insulin 2012-1

Question 4 LOA: 1 **INSULIN**	Describe the different types of insulin used in the routine management of Type I Diabetes. *Prompt: Please describe in terms of duration of action*	**Rapid and short acting** Clear soln, neutral pH, contain Zn rapid onset, short duration e.g. insulin neutral, insulin lispro, insulin glulusine - **Intermediate acting** Turbid soln, neutral pH, protamine in phosphate buffer (NPH) to prolong action e.g. insulin isophane, insulin aspart protamine **Long acting** Clear solution, soluble Slow onset, prolonged action Daily admin mimics basal insulin secretion e.g. insulin glargine, insuline detemir	Pass criteria: Identify existence of rapid, intermediate and long-acting insulin Aware that combination of therapies required to cover both basal requirements and post-prandial periods
	How are these properties used to achieve optimum glycaemic control?	Tight glycaemic control is achieved by a combination of insulins with different durations of action with an aim of replacing the basal insulin requirements (50%) and meal requirements (50%). This is done with **combinations of insulins with different duration of actions**	
	What type of insulin is used for intravenous infusion and why?	Short-acting regular soluble insulin as it immediately dissociates on dilution and so is able to more precisely delivered.	
	Optional: Describe the principles of operation of a subcutaneous insulin infusion device. PROMPT: Insulin pump.	External open-loop pump for insulin delivery. Delivers individualised basal and bolus insulin replacement doses based on blood glucose monitoring. Programmed by user. Consists of insulin reservoir, program chip, keypad and display screen attached to subcutaneously inserted infusion set.	

Insulin 2004-2

Insulin	Describe the action of insulin on the liver What are the complications of insulin therapy ?	Anabolic, anti catabolic 2 of immune,hypoglycaemia, lipodystrophy, immune resistance	

Metformin 2014-2-A

Stem: We will now move onto Pharmacology. He is a diabetic on metformin.			
Question 4 Metformin (p 757) **Subject**: Pharm LOA: 1	1. Describe the pharmacokinetics of metformin	Well absorbed, not protein bound, not metabolised, elimination half-life 1.5 to 3 hours **Excreted by kidney as unchanged compound.**	Bold and one other to pass.
	2. Outline some common side effects of metformin	**GI most common (20%) – limits compliance with this drug.** HAGMA **(lactic acidosis)** esp in patients with coexistent renal disease, EtOH, chronic cardiopulmonary disease.	Bold to pass.
	3. Contrast the mechanism of action of metformin (biguanide) and glipizide (sulfonylurea).	Glipizide – **Increases insulin release from pancreas** (patients more prone to hypoglycaemia with glipizide compared with metformin) Decreases serum glucagon levels Metformin Mechanism unclear but: May reduce hepatic gluconeogenesis. **Not dependent on functioning pancreatic B cells – so doesn't influence insulin release from pancreas** May directly simulate glycolysis in tissues with increased glucose removal from blood Decreases glucose absorption in the gut	Bold to pass.

Octreotide 2017-1-C

Stem: His treatment includes the administration of Octreotide. Moving on to Pharmacology.			
Question 5 Octreotide **Subject**: Pharm LOA 2	a) What is the mechanism of action of Octreotide? b) What are the routes of administration for octreotide? c) What are its adverse effects?	Somatostatin analog, **reduces** splanchnic & **portal blood flow** by poorly understood mechanism & hence variceal pressures. Inhibits endocrine & paracrine factor secretion including insulin, glucagon, gastrin, GH, TSH, IV, IM, SC Anaphylaxis, Local irritation during injection (redness, burning) GIT symptoms (nausea & vomiting, decreased intestinal motility, bowel obstruction, cholelithiasis) Hypo/hyper glycaemia Cardiac – sinus bradycardia, conduction disturbances	Bold plus general concept Any 2 to pass Any 2 to pass

Octreotide 2016-1-B

Stem: Moving onto Pharmacology. He is treated with octreotide.			
Question 4 Octreotide – mechanism of action, pharmacokinetics. LOA: 2	*a. Explain the mechanism of action of octreotide.* *b. Describe the pharmacokinetics of octreotide.* *c. What are some of its clinical uses?* *Bonus: What are its adverse effects?"*	A somatostatin analogue that inhibits the release of GH, TSH, glucagon**, insulin and gastrin.** **[Reduces splanchnic blood flow / portal pressure].** Plasma elimination half-life is 80 minutes. Metabolised by liver (30-40%) & 20% excreted unchanged by kidney. Acute control of **bleeding from oesophageal varices,** sulphonylurea overdose, reduce symptoms caused by hormone secreting tumours eg: acromegaly, carcinoid, gastrinoma, locating endocrine tumours using radiolabelled octreotide. Side effects include nausea, vomiting, abdo cramps, flatulence, steatorrhoea.	2 bold Know $T_{1/2}$ (range 40-120 min) Bold plus 1 1

Octreotide 2013-1

Question 5 **OCTREOTIDE** LOA: 2	What are the therapeutic uses for octreotide?	Control of **bleeding gastro-oesophageal varices,** sulphonylurea induced hypoglycaemia, pituitary and carcinoid tumors.	**Bold to pass**
	What is the mechanism of action of octreotide in acute variceal bleeding?	**Reduces splanchnic blood flow/portal venous pressure.** Exact mechanism of how this occurs is not known.	**Bold to pass**
	How is it administered in acute variceal bleeding?	**IV bolus and infusion** (50mcg bolus then 25-50mcg/hr) or SC	**Bold to pass**
	Why is an infusion required?	**Short half-life**	**Bold to pass**

Octreotide 2008-1

Octreotide	Explain the rationale for the use of octreotide in upper gastrointestinal bleeding What are the pharmacokinetic differences between octreotide and somatostatin? (Supp Question – What other agents may be useful in the prevention and treatment of upper GI bleeding)	Octreotide reduces splanchnic blood flow, (? By glucagon release inhibition) therefore reduces portal venous pressure. This reduces blood loss from bleeding oesophageal varices and in some cases of severe duodenal ulcer related bleeding. Octreotide is a somatostatin analogue that has a longer half life than somatostatin (1.5hrs vs 3 min) so can be given as an IV infusion or subcutaneously. (Pass – reduces splanchnic blood flow)	

Oral hypoglycaemics 2012-2

			poor renal function!
Question 2 Oral hypoglycaemics LOA: 1	Describe the pharmacokinetics of metformin? What are the side effects of metformin? With regard to sulphonylureas, what is the mechanism of action of glipizide? (prompt: it's a sulphonylurea)	Well absorbed, not protein bound, not metabolised, elimination t1/2: 1.5-3 hours, **excreted by kidney** as unchanged compound Gastrointestinal most common 20%, decreased absorption Vit B12, **lactic acidosis** esp with renal disease, ETOH, chronic cardiopulmonary disease **Increase insulin release from the pancreas** bind to receptor associated with ATP sensitive K channel, inhibits efflux of K ions, results in depolarization and opens ca channel, influx of Ca causes release of preformed insulin Reduction of serum glucagon levels Closure of potassium channels in extrapancreatic tissues	Bold Bold Patients more prone to hypo than with biguanides eg metformin

Oral hypoglycaemics 2003-2

Pharm Session 2 Topics	Questions	Essential knowledge	Comments	Pass / Fail (Score)
Oral hypoglycaemic agents	1. Regarding sulphonylureas and bigaunides, compare their mechanisms of action.	**Sulphonylureas increase insulin release.** Act via a specific receptor which causes an increase in intracellular Ca, which triggers insulin release. There are also receptors in cells on binding proteins in secretory granules, which may cause a direct action on exocytosis of insulin. Other peripheral effects may be to reduce serum glucagon and potentiate insulin effects on cells. **Biguanides** are 'euglycaemic agents". They **do not require functional islet cells to reduce blood sugar.** Their possible actions are to directly stimulate glycolysis in tissues and blood; reduce hepatic gluconeogenesis; reduce GIT absorption; reduce plasma glucagon.	**Bold items required to pass with some additional explanation.**	
	2. How do the major side effects of the two groups of drugs differ?	**Biguanides can cause lactic acidosis.** They reduce gluconeogenesis and reduce lactic acid uptake in the liver. More likely in patients with renal disease, alcoholism, liver disease and chronic tissue hypoxia. **Sulphonylureas more commonly cause hypoglycaemia.** More likely in the elderly and with drugs with long t ½ e.g. chlorpropamide.	**Bold items required to pass with some additional explanation.**	

Oral hypoglycaemics 2003-1

QUEST 5. Oral hypoglycemic agents

FIRST QUESTION	What are the different types of oral antidiabetic agents ?	
	a. Insulin secretagogues (**sulfonylureas,** meglitinides) **b. Biguanides** c. Thiazolidinediones – enhance target tissue insulin sensitivity d. Alpha-glucosidase inhibitors – competitive inhibitors of intestinal alpha glucosidases – defers digestion to distal small intestine	
SECOND QUESTION	What is the mechanism of action of the sulfonylureas	
	- increase insulin release from pancreas - reduce serum glucagons levels - extrapancreatic effect to potentiate action of insulin on target cells (last 2 ?clin.sig.)	
THIRD QUESTION	How do the biguanides differ from the sulfonylurease in their action	
	Not need functioning pancreatic B cells - direct simulation of glycolysis in tissue - reduce hepatic gluconeogensis - slowing glc.absorption from GI tract - reduction of plasma glucagons levels	
FOURTH QUESTION	What are the clinical advantages of the different oral antidiabetic agents?	
	a. Biguanides = **Refractory obesity** where insulin resistance b. Combination with sulfonylureas in Type II Diabetes c. Newer sulfonylureas are liver metabolized so can be used in renal failure	

Sulphonylureas 2016-2-A

Stem: A 46-year-old man is referred by his GP with poorly controlled diabetes. Starting with Pharmacology. He is on Gliclazide			
TOPIC	QUESTIONS	KNOWLEDGE **(essential in bold)**	NOTES
Question 1 Gliclazide (Pharmacodynamics / kinetics) **Subject:** Pharm LOA: 1	1) What type (class) of drug is Gliclazide?	**A sulphonylurea.**	Bold to pass
	2) Describe the mechanism of action of the sulfonylureas? *Prompt: What ion channels are involved?*	**They increase the release of insulin from the pancreas** (specifically from pancreatic beta cells). They bind to a receptor -> inhibition of efflux of K^+ ions through a linked ATP-sensitive potassium channel -> (extracellular) depolarization. The depolarization opens a voltage-gated calcium channel -> Ca influx -> release of preformed insulin. Long term use also -> reduced serum glucagon levels. Mechanism for effect unclear, but may involve indirect inhibition due to enhanced release of insulin and somatostatin which inhibit alpha-cell secretion.	To pass bold + concept of ion channels
	3) What are the pharmacokinetics of the sulfonylureas?	**Hepatically metabolized** to products which are inactive or have very low activity. **Renally** excreted. Variable (but moderate) T ½ (Gliclazide 8hrs, Glimepiride 12-24hrs, Glipizide 12-24hrs)	Bold to pass

Sulphonylureas 2015-1-C

Stem: Moving onto Pharmacology. He has been on Gliclazide for his Diabetes			
Question 4 Sulfonyl Ureas **Subject:** Pharm LOA: 1	1. What class of drug is gliclazide? 2. Describe the mechanism of action of sulfonylureas. 3. What are the pharmacokinetic properties of gliclazide? 4. What are potential adverse effects of gliclazide?	1. **Sulphonylurea** 2. **Stimulates insulin secretion from functional pancreatic beta cells** • Binding of sulphonylurea to receptor inhibits potassium efflux causing extracellular depolarisation • Results in opening of voltage gated calcium channels • Calcium influx causes release of preformed insulin 3. Administered orally – good oral bioavailability (80%) Protein bound – volume of distribution ~ 20L **Hepatic metabolism** to inactive metabolites **Half life approx. 12 hours** Predominantly renally excreted (80%) 4. **Hypoglycaemia** **GI upset** – nausea, vomiting, abdominal pain, diarrhoea Rash/pruritis	Bold to pass Bold Bold Hypoglycaemia plus one

Sulphonylureas 2008-2

Question 4: Sulfonylureas	1. What are the mechanisms of action of the Sulfonylureas? Prompt: How do sulphonylureas lower glucose? Describe another mechanism? 3. What are the adverse effects of sulfonylurea therapy?	Increased secretion of insulin - Bind to pancreatic B cell receptor causing increased release of Insulin -Reduced serum glucagon levels – with chronic use thought to be due to indirect inhib effects of insulin and somatostatin on a cells -Potentiation of insulin action on target tissues – increased binding of insulin to tissue receptors ?due to indirect effect of reduced glycemia or FFA levels Prolonged hypoglycemia; Alcohol intolerance – flushing; Dilutional hyponatremia (genetic predisposition) Jaundice, Leucopenia, thrombocytopenia (Chlorpropamide)	Bind to B cell; 1 of other 2 Hypoglycaemia