

**REPORT ON STUDENT INDUSTRIAL WORK EXPERIENCE SCHEME (SIWES) AT IRRUA SPECIALIST TEACHING HOSPITAL , EDO STATE BY**

EHIKIOYA SANDRA

**MATRIC NUMBER: 16/MHS07/010**

**COLLEGE OF MEDICINE AND HEALTH SCIENCE**

**DEPARTMENT OF PHARMACOLOGY AND THERAPEUTICS**

**AFE BABALOLA UNIVERSITY, ADO EKITI, EKITI STATE- NIGERIA.**

**DEDICATION**

This report is dedicated to my family, friends, department of pharmacology and therapeutics and to my colleagues that worked with at my place of industral training that took place at IRRUA SPEACIALIST TEACHING HOSPITAL (I.S.T.H)

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**ABSTRACT**

The student industrial scheme (SIWES) is a scheme designed by the federal government to expose 200/ 300 level pharmacology students of teritary institution to aquisition of skills relevant to their course of study as well as industrial experience. The 2018/2019 SIWES was undertaken at IRRUA SPEACIALIST TEACHING HOSPITAL( I.S.T.H) i was accepted into their internship program available for students in teritary institution, I was placed in the department of PHARMACY at out-patient department(OPD). We carried out how to dispense drugs to patient of different disease condition e.g anti hypertensive drugs for hypertension, anti diabetic drugs for diabetes etc.

The scheme provided adequate exposure to practical work as well as acqusition of industrial techanical skills relevant to the course of study.

**INTRODUCTION & HISTORY OF SIWES**

The program was created to give students experience in addition to theoretical learning. The industrial training policy was introduced by federal government of Nigeria in 1973. This project was necessary to improve practical skills of students. SIWES has become a necessary pre condition of graduation.

The Federal Government of Nigeria created the Industrial Training Funds (ITF) in the year 1972 in an effect to boost the educational standard and industrial development of Nigeria.

ITF insisted that Students Industrial Work Experience Scheme (SIWES). This is accepted skill training program, which form part of the approved minimum academic standard in some different Degree, Diplomas and N.C.E. for Nigerian universities, polytechnics and co

The SIWES programmed expose students to the needed experience of training, such as handling machine and equipment.

It provide job opportunities for students whose graduate from universities, polytechnics and colleges of education, the effectiveness of this scheme gives the job specification in which technical lecturers, students and employers contribute after employed.

**HISTORY OF MY INDUSTRIAL TRAINING CENTER AT IRRUA SPECIALIST TEACHING HOSPITAL (I.S.T.H)**

Irrua specialist teaching hospital, commissioned in 1993 as a 230 bed hospital, is currently a 375 bed hospital. Its strategic location along the Benin- Abuja expressway in irrua, the headquarter of esan central LGA in edo central senatorial district. Enables it to serve the central and the northern senatorial district of edo state, as well as part of the southern senatorial district. In addition, it also receives patient from the neighboring state of delta, kogi and ondo, furthermore, it serves as the teaching hospital to Ambrose alli university Ekpoma.

The vision of the hospital is to become a center of excellence in medicine, particularly in the areas of rural and sub urban medicine and the diagnosis and the management of viral hemorrhagic fevers, especially Lassa fever.

The mission to provide qualitative, affordable, readily available, acceptable and functional health care services, and training in medicine and related fields in order to reduce morbidity and mortality in the line with the national health target

**AIMS AND OBJECTIVES OF SIWES**

1. To expose and prepare student for the industrial work situation, they are to meet after graduation.
2. To improve the relationship between educational institutions and industrial sectors.
3. To give the student chance of putting the theoretical part of what they learnt in the class into practice.
4. To expose the student to the practical aspect of what they learnt in the classroom
5. To give student opportunity to decide and appreciate this practical experience.

**DEFINITION OF ANTIHYPERTENSIVE DRUGS**

* Antihypertensive are a class of drugs that are used to treat hypertension (high blood pressure). Antihypertensive therapy seeks to prevent the complications of high blood pressure, such as stroke and myocardial infarction. Evidence suggests that reduction of the blood pressure by 5 mmHg can decrease the risk of stroke by 34%, of ischemic heart disease by 21%, and reduce the likelihood of dementia, heart failure, and mortality from cardiovascular disease.[There are many classes of antihypertensive, which lower blood pressure by different means. Among the most important and most widely used drugs are thiazide diuretics, calcium channel blockers, ACE inhibitors, angiotensin II receptor antagonists (ARBs), and beta blockers.

**CLASSES OF ANTIHYPERTENSIVE DRUGS**

* ACE inhibitors (Angiotensin-converting enzyme inhibitors)
* ARBs (Angiotensin II receptor blockers), also called Sartans.
* Beta blockers.
* Calcium channel blockers.
* Direct renin inhibitors.
* Diuretics.
* **ACE INHIBITOR**

An angiotensin-converting-enzyme inhibitor (ACE inhibitor) is a [pharmaceutical drug](https://en.m.wikipedia.org/wiki/Pharmaceutical_drug) used primarily for the treatment of [hypertension](https://en.m.wikipedia.org/wiki/Hypertension) (elevated blood pressure) and [congestive heart failure](https://en.m.wikipedia.org/wiki/Congestive_heart_failure).This group of drugs causes relaxation of blood vessels as well as a decrease in [blood volume](https://en.m.wikipedia.org/wiki/Blood_volume), which leads to lower [blood pressure](https://en.m.wikipedia.org/wiki/Blood_pressure) and decreased oxygen demand from the [heart](https://en.m.wikipedia.org/wiki/Heart). They [inhibit](https://en.m.wikipedia.org/wiki/Enzyme_inhibitor) the [angiotensin-converting enzyme](https://en.m.wikipedia.org/wiki/Angiotensin-converting_enzyme), an important component of the [renin–angiotensin system](https://en.m.wikipedia.org/wiki/Renin%E2%80%93angiotensin_system).

 Examples of ACE inhibitors include:

* Benazepril
* Captopril.
* Enalapril
* Fosinopril.
* Lisinopril
* Moexipril.
* Perindopril
* Quinapril

## **Medical use:**

ACE inhibitors were initially approved for the treatment of hypertension and can be used alone or in combination with other anti-hypertensive medications. Later, they were found useful for other cardiovascular and kidney diseases including:

* Acute [myocardial infarction](https://en.m.wikipedia.org/wiki/Myocardial_infarction) (heart attack)
* [Heart failure](https://en.m.wikipedia.org/wiki/Heart_failure) (left ventricular systolic dysfunction)
* Kidney complications of [diabetes mellitus](https://en.m.wikipedia.org/wiki/Diabetes_mellitus) ([diabetic nephropathy](https://en.m.wikipedia.org/wiki/Diabetic_nephropathy)) In treating high blood pressure, ACE inhibitors are often the first drug choice, particularly when diabetes is present, but age can lead to different choices and it is common to need more than one drug to obtain the desired improvement. There are fixed-dose [combination drugs](https://en.m.wikipedia.org/wiki/Combination_drugs), such as [ACE inhibitor and thiazide combinations](https://en.m.wikipedia.org/wiki/ACE_inhibitor_and_thiazide_combination). ACE inhibitors have also been used in [chronic kidney failure](https://en.m.wikipedia.org/wiki/Chronic_kidney_failure) and kidney involvement in [systemic sclerosis](https://en.m.wikipedia.org/wiki/Systemic_sclerosis) (hardening of tissues, as scleroderma renal crisis). In those with stable coronary artery disease, but no heart failure, benefits are similar to other usual treatments.

## **Mechanism of action:**

ACE inhibitors reduce **t**he activity of the renin–angiotensin–aldosterone system (RAAS) as the primary etiologic (causal) event in the development of hypertension in people with diabetes mellitus, as part of the insulin-resistance syndrome or as a manifestation of renal disease.

### Renin–angiotensin–aldosterone system

Renin–angiotensin–aldosterone system is a major blood pressure regulating mechanism. Markers of electrolyte and water imbalance in the body such as [hypotension](https://en.m.wikipedia.org/wiki/Hypotension), low [distal tubule](https://en.m.wikipedia.org/wiki/Distal_convoluted_tubule) [sodium](https://en.m.wikipedia.org/wiki/Sodium_in_biology) concentration, decreased blood volume and high [sympathetic](https://en.m.wikipedia.org/wiki/Sympathetic_nervous_system) tone trigger the release of the enzyme [renin](https://en.m.wikipedia.org/wiki/Renin) from the cells of [juxtaglomerular apparatus](https://en.m.wikipedia.org/wiki/Juxtaglomerular_apparatus) in the kidney. Renin activates a circulating liver derived prohormone [angiotensinogen](https://en.m.wikipedia.org/wiki/Angiotensin) by proteolytic cleavage of all but its first ten [amino acid](https://en.m.wikipedia.org/wiki/Amino_acid) residues known as [angiotensin I](https://en.m.wikipedia.org/wiki/Angiotensin_I). [ACE](https://en.m.wikipedia.org/wiki/Angiotensin_converting_enzyme) (Angiotensin converting enzyme) then removes a further two residues, converting angiotensin I into [angiotensin II](https://en.m.wikipedia.org/wiki/Angiotensin_II). [ACE](https://en.m.wikipedia.org/wiki/Angiotensin_converting_enzyme) is found in the [pulmonary circulation](https://en.m.wikipedia.org/wiki/Pulmonary_circulation) and in the [endothelium](https://en.m.wikipedia.org/wiki/Endothelium) of many blood vessels. The system increases blood pressure by increasing the amount of salt and water the body retains, although angiotensin is also very good at causing the blood vessels to tighten (a potent [vasoconstrictor](https://en.m.wikipedia.org/wiki/Vasoconstrictor))

### **Effects:**

ACE inhibitors block the conversion of Angiotensin I (ATI) to Angiotensin II (ATII). They thereby lower [arteriolar](https://en.m.wikipedia.org/wiki/Arteriole) resistance and increase venous capacity; decrease [cardiac output](https://en.m.wikipedia.org/wiki/Cardiac_output), [cardiac index](https://en.m.wikipedia.org/wiki/Cardiac_index), stroke work, and [volume](https://en.m.wikipedia.org/wiki/Stroke_volume); lower resistance in blood vessels in the kidneys; and lead to increased [natriuresis](https://en.m.wikipedia.org/wiki/Natriuresis) (excretion of sodium in the urine). Renin increases in concentration in the blood as a result of negative feedback of conversion of ATI to ATII. ATI increases for the same reason; ATII and aldosterone decrease. [Bradykinin](https://en.m.wikipedia.org/wiki/Bradykinin) increases because of less inactivation by ACE.

Under normal conditions, angiotensin II has these effects:

* Vasoconstriction (narrowing of blood vessels) and vascular smooth muscle hypertrophy (enlargement) induced by ATII may lead to increased blood pressure and hypertension. Further, constriction of the [efferent arterioles](https://en.m.wikipedia.org/wiki/Efferent_arteriole) of the kidney leads to increased perfusion pressure in the [glomeruli](https://en.m.wikipedia.org/wiki/Glomerulus).
* It contributes to [ventricular remodeling](https://en.m.wikipedia.org/wiki/Ventricular_remodeling) and [ventricular hypertrophy](https://en.m.wikipedia.org/wiki/Ventricular_hypertrophy) of the heart through stimulation of the [proto-oncogenes](https://en.m.wikipedia.org/wiki/Proto-oncogene) [c-fos](https://en.m.wikipedia.org/wiki/C-fos), [c-jun](https://en.m.wikipedia.org/wiki/C-jun), [c-myc](https://en.m.wikipedia.org/wiki/C-myc), [transforming growth factor beta](https://en.m.wikipedia.org/wiki/Transforming_growth_factor_beta) (TGF-B), through fibrogenesis and apoptosis (programmed cell death).
* Stimulation by ATII of the [adrenal cortex](https://en.m.wikipedia.org/wiki/Adrenal_cortex) to release [aldosterone](https://en.m.wikipedia.org/wiki/Aldosterone), a hormone that acts on kidney tubules, causes sodium and chloride ions retention and potassium excretion. Sodium is a "water-holding" ion, so water is also retained, which leads to increased blood volume, hence an increase in blood pressure.
* Stimulation of the posterior pituitary to release [vasopressin](https://en.m.wikipedia.org/wiki/Vasopressin) (antidiuretic hormone, ADH) also acts on the kidneys to increase water retention. If ADH production is excessive in heart failure, Na+ level in the plasma may fall (hyponatremia), and this is a sign of increased risk of death in heart failure patients.
* A decrease renal protein kinase

## **Adverse effects:**

Common [adverse drug reactions](https://en.m.wikipedia.org/wiki/Adverse_drug_reaction) include: hypotension, [cough](https://en.m.wikipedia.org/wiki/Cough), [hyperkalemia](https://en.m.wikipedia.org/wiki/Hyperkalemia), [headache](https://en.m.wikipedia.org/wiki/Headache), [dizziness](https://en.m.wikipedia.org/wiki/Vertigo_%28medical%29), [fatigue](https://en.m.wikipedia.org/wiki/Fatigue_%28physical%29), [nausea](https://en.m.wikipedia.org/wiki/Nausea), and [renal](https://en.m.wikipedia.org/wiki/Kidney)impairment. ACE inhibitors might increase [inflammation](https://en.m.wikipedia.org/wiki/Inflammation)-related [pain](https://en.m.wikipedia.org/wiki/Pain), perhaps mediated by the buildup of [bradykinin](https://en.m.wikipedia.org/wiki/Bradykinin) that accompanies ACE inhibition.

* **ANGIOTENSIN II RECEPTOR BLOCKERS**

Angiotensin II receptor blockers (ARBs), also known as angiotensin II receptor antagonists, AT1 receptor antagonists or sartans, are a group of pharmaceuticals that modulate the [renin–angiotensin system](https://en.m.wikipedia.org/wiki/Renin%E2%80%93angiotensin_system). Their main uses are in the treatment of [hypertension](https://en.m.wikipedia.org/wiki/Hypertension) (high blood pressure), [diabetic nephropathy](https://en.m.wikipedia.org/wiki/Diabetic_nephropathy) ([kidney damage](https://en.m.wikipedia.org/wiki/Kidney_damage) due to [diabetes](https://en.m.wikipedia.org/wiki/Diabetes_mellitus)) and [congestive heart failure](https://en.m.wikipedia.org/wiki/Congestive_heart_failure). They *selectively* [block](https://en.m.wikipedia.org/wiki/Receptor_antagonist) the activation of [AT1 receptors](https://en.m.wikipedia.org/wiki/Angiotensin_II_receptor_type_1), preventing the [binding](https://en.m.wikipedia.org/wiki/Ligand_%28biochemistry%29) of [angiotensin II](https://en.m.wikipedia.org/wiki/Angiotensin#Angiotensin_II) compared to [ACE inhibitors](https://en.m.wikipedia.org/wiki/ACE_inhibitors).

ARBs and the similar-attributed ACE inhibitors are both indicated as the first-line [antihypertensives](https://en.m.wikipedia.org/wiki/Antihypertensive) in patients developing [hypertension](https://en.m.wikipedia.org/wiki/Hypertension) along the [left-sided](https://en.m.wikipedia.org/wiki/Left_ventricular)[heart failure](https://en.m.wikipedia.org/wiki/Heart_failure) However, ARBs appear to produce less adverse effects compared to ACEs.

Examples of angiotensin II receptor blockers include:

* Eprosartan.
* Azilsartan
* Candesartan
* Irbesartan
* Losartan
* Olmesartan
* Telmisartan
* Valsartan

## **Mechanism of action:**

These substances are AT1-receptor antagonists; that is, they block the activation of [angiotensin II AT1receptors](https://en.m.wikipedia.org/wiki/Angiotensin_receptor). AT1 receptors are found in [smooth muscle](https://en.m.wikipedia.org/wiki/Smooth_muscle)cells of vessels, cortical cells of the [adrenal gland](https://en.m.wikipedia.org/wiki/Adrenal_gland), and [adrenergic](https://en.m.wikipedia.org/wiki/Adrenaline) nerve synapses. Blockage of AT1receptors directly causes [vasodilation](https://en.m.wikipedia.org/wiki/Vasodilation), reduces secretion of [vasopressin](https://en.m.wikipedia.org/wiki/Vasopressin), and reduces production and secretion of [aldosterone](https://en.m.wikipedia.org/wiki/Aldosterone), among other actions. The combined effect reduces blood pressure.

The specific efficacy of each ARB within this class depends upon a combination of three [pharmacodynamic](https://en.m.wikipedia.org/wiki/Pharmacodynamic) (PD) and [pharmacokinetic](https://en.m.wikipedia.org/wiki/Pharmacokinetic) (PK) parameters. Efficacy requires three key PD/PK areas at an effective level; the parameters of the three characteristics will need to be compiled into a table similar to one below, eliminating duplications and arriving at consensus values; the latter are at variance now.

## **Adverse effects:**

This class of drugs is usually well tolerated. Common [adverse drug reactions](https://en.m.wikipedia.org/wiki/Adverse_drug_reaction) (ADRs) include: dizziness, headache, and/or [hyperkalemia](https://en.m.wikipedia.org/wiki/Hyperkalemia). Infrequent ADRs associated with therapy include: first dose [orthostatic hypotension](https://en.m.wikipedia.org/wiki/Orthostatic_hypotension), rash, diarrhea, [dyspepsia](https://en.m.wikipedia.org/wiki/Dyspepsia), abnormal liver function, muscle cramp, [myalgia](https://en.m.wikipedia.org/wiki/Myalgia), back pain, [insomnia](https://en.m.wikipedia.org/wiki/Insomnia), decreased [hemoglobin](https://en.m.wikipedia.org/wiki/Hemoglobin) levels, [renal impairment](https://en.m.wikipedia.org/wiki/Renal_impairment), [pharyngitis](https://en.m.wikipedia.org/wiki/Pharyngitis), and/or nasal congestion.

While one of the main rationales for the use of this class is the avoidance of dry cough and/or angioedema associated with ACE inhibitor therapy, rarely they may still occur. In addition, there is also a small risk of cross-reactivity in patients having experienced [angioedema](https://en.m.wikipedia.org/wiki/Angioedema) with ACE inhibitor therapy.

## **BETA-BLOCKERS**

Beta-blockers are a class of medication used to block the effects of stress hormones such as adrenaline on the heart. They’re often prescribed for irregular heartbeats, high blood pressure, and after heart attacks.

Less commonly, beta-blockers may be used to treat:

* [glaucoma](https://www.healthline.com/health/glaucoma)
* [migraines](https://www.healthline.com/health/migraine-treatments)
* [anxiety disorders](https://www.healthline.com/health/anxiety-drugs)
* [hyperthyroidism](https://www.healthline.com/health/hyperthyroidism)
* [tremors](https://www.healthline.com/symptom/tremor)

Doctors typically turn to beta-blockers for [high blood pressure](https://www.healthline.com/health/high-blood-pressure-hypertension-treatment) when other medications, such as [diuretics](https://www.healthline.com/health/diuretics), aren’t working or have too many side effects. They may be used in combination with other blood pressure-lowering medications, including [ACE inhibitors](https://www.healthline.com/health/heart-disease/ACE-inhibitors) and [calcium channel blockers](https://www.healthline.com/health/heart-disease/calcium-channel-blockers).

**Mechanism of action:**

Because of the way they work in the body, beta-blockers are also called beta-adrenergic blocking substances.

Different types of beta-blockers work differently. In general, these medications enhance the heart’s ability to relax. Your heart will beat slower and less forcefully when beta-blockers are working. This can help [reduce blood pressure](https://www.healthline.com/health/high-blood-pressure-hypertension/lower-it-fast) and alleviate [irregular heart rhythms](https://www.healthline.com/symptom/abnormal-heart-rhythms).

Some beta-blockers only work on the heart itself, while others affect the heart and blood vessels.

Your doctor may prescribe beta-blockers even if you have few symptoms of [heart problems](https://www.healthline.com/health/heart-disease) or [heart failure](https://www.healthline.com/health/heart-failure). These medications can actually improve the heart’s ability to beat.

Commonly prescribed beta-blockers include:

* [acebutolol](https://www.healthline.com/health/acebutolol-oral-capsule)
* [atenolol](https://www.healthline.com/health/atenolol/oral-tablet)
* [bisoprolol](https://www.healthline.com/health/bisoprolol-oral-tablet)
* carteolol
* esmolol
* [metoprolol](https://www.healthline.com/health/metoprolol-oral-tablet)
* [nadolol](https://www.healthline.com/health/nadolol-oral-tablet)
* [nebivolol](https://www.healthline.com/health/nebivolol-oral-tablet)
* [propranolol](https://www.healthline.com/health/propranolol/oral-tablet)

## **Side effects and risks of beta-blockers:**

People with [asthma](https://www.healthline.com/health/asthma) typically shouldn’t take beta-blockers since they can [trigger](https://www.healthline.com/health/asthma-causes) asthma attacks.

Because beta-blockers may affect the control of [blood sugar](https://www.healthline.com/health/glucose), they’re not usually recommended for people with [diabetes](https://www.healthline.com/health/diabetes/beta-blockers-what-you-need-to-know).

Side effects of these medications can vary. Many people will experience:

* [fatigue](https://www.healthline.com/symptom/fatigue), cold hands, [headache](https://www.healthline.com/health/headache), digestive problems, [constipation](https://www.healthline.com/symptom/constipation), [diarrhea](https://www.healthline.com/symptom/diarrhea), [dizziness](https://www.healthline.com/symptom/dizziness)

Rarely, you may experience:

* [shortness of breath](https://www.healthline.com/symptom/shortness-of-breath), [trouble sleeping](https://www.healthline.com/symptom/difficulty-sleeping), decreased libido, [depression](https://www.healthline.com/health/depression)
* **CALCIUM CHANNEL BLOCKERS**

Calcium channel blockers (CCB), calcium channel antagonists or calcium antagonists are several medications that disrupt the movement of [calcium](https://en.m.wikipedia.org/wiki/Calcium)(Ca2+) through [calcium channels](https://en.m.wikipedia.org/wiki/Calcium_channel). Calcium channel blockers are used as [antihypertensive drugs](https://en.m.wikipedia.org/wiki/Antihypertensive_drug), i.e., as medications to decrease [blood pressure](https://en.m.wikipedia.org/wiki/Blood_pressure) in patients with [hypertension](https://en.m.wikipedia.org/wiki/Hypertension). CCBs are particularly effective against large vessel stiffness, one of the common causes of elevated [systolic](https://en.m.wikipedia.org/wiki/Systolic) blood pressure in [elderly patients](https://en.m.wikipedia.org/wiki/Elderly_care). Calcium channel blockers are also frequently used to alter [heart rate](https://en.m.wikipedia.org/wiki/Heart_rate), to prevent [cerebral vasospasm](https://en.m.wikipedia.org/wiki/Cerebral_vasospasm), and to reduce [chest pain](https://en.m.wikipedia.org/wiki/Chest_pain) caused by [angina pectoris](https://en.m.wikipedia.org/wiki/Angina_pectoris).

Examples of calcium channel blockers include:

* Amlodipine
* Diltiazem
* Felodipine
* Isradipine
* Nicardipine
* Nifedipine
* Nisoldipine

Verapamil acting on the muscle cells in the arterial walls

**Mechanism of action :**

 In the body's tissues, the concentration of calcium ions (Ca2+) outside cells is normally about 10000-fold higher than the concentration inside cells. Embedded in the [membrane](https://en.m.wikipedia.org/wiki/Cell_membrane) of some cells are [calcium channels](https://en.m.wikipedia.org/wiki/Calcium_channel). When these cells receive a certain signal, the channels open, letting calcium rush into the cell. The resulting increase in intracellular calcium has different effects in different types of cells. Calcium channel blockers prevent or reduce the opening of these channels and thereby reduce these effects.

Several types of calcium channels occur, with a number of classes of blockers, but almost all of them preferentially or exclusively block the [L-type](https://en.m.wikipedia.org/wiki/L-type_calcium_channel) voltage-gated calcium channel.

[Voltage-dependent calcium channels](https://en.m.wikipedia.org/wiki/Voltage-dependent_calcium_channel) are responsible for excitation-[contraction](https://en.m.wikipedia.org/wiki/Muscle_contraction) coupling of [skeletal](https://en.m.wikipedia.org/wiki/Skeletal_muscle), [smooth](https://en.m.wikipedia.org/wiki/Smooth_muscle), and [cardiac muscle](https://en.m.wikipedia.org/wiki/Cardiac_muscle) and for regulating [aldosterone](https://en.m.wikipedia.org/wiki/Aldosterone) and [cortisol](https://en.m.wikipedia.org/wiki/Cortisol) secretion in [endocrine cells](https://en.m.wikipedia.org/wiki/Endocrine_system)of the [adrenal cortex](https://en.m.wikipedia.org/wiki/Adrenal_cortex). In the heart, they are also involved in the conduction of the [pacemaker](https://en.m.wikipedia.org/wiki/Cardiac_pacemaker) signals. CCBs used as medications primarily have four effects:

* By acting on [vascular smooth muscle](https://en.m.wikipedia.org/wiki/Vascular_smooth_muscle), they reduce contraction of the arteries and cause an increase in [arterial](https://en.m.wikipedia.org/wiki/Artery) diameter, a phenomenon called [vasodilation](https://en.m.wikipedia.org/wiki/Vasodilation) (CCBs do not work on [venous](https://en.m.wikipedia.org/wiki/Vein) smooth muscle).
* By acting on cardiac muscles ([myocardium](https://en.m.wikipedia.org/wiki/Myocardium)), they reduce the force of contraction of the heart.
* By slowing down the conduction of electrical activity within the heart, they slow down the heart beat.
* By blocking the calcium signal on adrenal cortex cells, they directly reduce aldosterone production, which correlates to lower blood pressure.

**Side effects of this drug include but are not limited to:**

* [Constipation](https://en.m.wikipedia.org/wiki/Constipation)
* Dizziness, headache, redness in the face
* Fluid buildup in the legs and ankle [edema](https://en.m.wikipedia.org/wiki/Edema)
* [Gingival overgrowth](https://en.m.wikipedia.org/wiki/Gingival_enlargement)
* Rapid heart rate
* Slow heart rate
* **RENIN INHIBITORS**

Renin inhibitors are a group of [pharmaceutical drugs](https://en.m.wikipedia.org/wiki/Pharmaceutical_drugs)used primarily in treatment of [essential](https://en.m.wikipedia.org/wiki/Essential_hypertension) [hypertension](https://en.m.wikipedia.org/wiki/Hypertension)(high blood pressure). These drugs [inhibit](https://en.m.wikipedia.org/wiki/Enzyme_inhibitor) the first and [rate-limiting step](https://en.m.wikipedia.org/wiki/Rate-determining_step) of the [renin–angiotensin–aldosterone system](https://en.m.wikipedia.org/wiki/Renin%E2%80%93angiotensin%E2%80%93aldosterone_system) (RAAS), namely the conversion of [angiotensinogen](https://en.m.wikipedia.org/wiki/Angiotensinogen) to [angiotensin I](https://en.m.wikipedia.org/wiki/Angiotensin_I). This leads to a totality in absence of Angiotensin II based on the rationale that renin only acts to inhibit this step unlike Angiotensin Converting

Examples of the drugs include:

* Amlodipine
* Hydrochlorothiazide
* Valsartan

**Mechanism of action:**

Renin inhibitors bind to the [active site](https://en.m.wikipedia.org/wiki/Active_site) of renin and inhibit the binding of renin to angiotensinogen, which is the rate-determining step of the RAAS cascade. Consequently, renin inhibitors prevent the formation of Ang I and Ang II. Renin inhibitors may also prevent Ang-(1-7), Ang-(1-9) and Ang-(1-5) formation, although it is not known if this is clinically important. Renin is highly selective for its only naturally occurring [substrate](https://en.m.wikipedia.org/wiki/Enzyme_substrate_%28biology%29) which is angiotensinogen, and the incidence of unwanted side effects with a renin inhibitor is infrequent and similar to [angiotensin II receptor antagonists](https://en.m.wikipedia.org/wiki/Angiotensin_II_receptor_antagonist). Ang II also functions within the RAAS as a negative feedback to suppress further release of renin. A reduction in Ang II levels or blockade of angiotensin receptors will suppress the feedback loop and lead to increased plasma renin concentrations (PRC) and [plasma renin activity](https://en.m.wikipedia.org/wiki/Plasma_renin_activity)(PRA). This can be problematic for [ACE inhibitor](https://en.m.wikipedia.org/wiki/ACE_inhibitor) and [angiotensin II receptor antagonist](https://en.m.wikipedia.org/wiki/Angiotensin_II_receptor_antagonist) therapy since increased PRA could partially overcome the pharmacologic inhibition of the RAAS cascade. Because renin inhibitors directly affect renin activity, decrease of PRA despite the increased PRC (from loss of the negative feedback) may be clinically advantageous.

* **DIURETICS DRUGS**

Diuretics, also called water pills, are medications designed to increase the amount of water and salt expelled from the body as urine. There are three types of prescription diuretics. They're often prescribed to help treat high blood diuretics: bumetanide. Ethacrynic acid. Furosemide. Torsemide.

* Thiazide diuretics: epitizide, Hydrochlorothiazide and chlorothiazide, , methyclothiazide.
* Loop diuretics, such as furosemide, torsemide and ethacrynic acid
* Potassium-sparing diuretics: amiloride. triamterene. spironolactone. eplerenone.ood pressure, but they're used for other conditions as well.

**RECOMMENDATION**

I hope with the implication of my industrial training, there will be no more anomalous feelings when the students started working after they have finished their course later .

**CONCLUSION**

In conclusion**,** the industrial training that I had already gone through for 13 weeks at irrua teaching hospital in the department of pharmacy. During my industrial training, there are many changes from the point of learning environments and discussion among colleagues. It can directly increase the dedication and rational attitude towards myself.

**EHIKIOYA SANDRA**

**400L**

**DEPARTMENT**

**OF PHARMACOLOGY AND THRAPEUTIC**