Introduction
Hypertension is a multifactor disease characterized by chronic elevation in blood pressure to levels equal to or above 140 mmHg systolic blood pressure (SBP) and above 90 mmHg of diastolic blood pressure (DBP). Considered a worldwide epidemic disease, hypertension is the main risk factor for cardiovascular disease, being epidemiologically closely associated with metabolic diseases such as obesity and diabetes. Hypertension is the main risk factor for cardiovascular diseases, which include stroke, coronary artery disease (CAD), and heart failure (HF) leading to 1.8 million deaths worldwide every year, cardiovascular disease leads to 17 millions of death per year, and, from this total, it is reported that high blood pressure is estimated to cause more than half of these deaths (over 9 million deaths every year), making it also the main risk factor in
The global disease burden. Aging is a major risk factor for developing hypertension. The prevalence of the disease increases with age, with a higher rate in men than women. People who don’t have hypertension at age 55 have 90% chance of developing it later in life.

The goal of hypertension treatment is to prevent death and complication by achieving and maintaining the blood pressure at 140/90 mm Hg or lower.

Life style modification is the first line of intervention for all patients with hypertension, but pharmacological is the cornerstone for the disease treatment to reduce the high blood pressure and prevent complications such as cardiovascular and renal morbidity and mortality.

For many years the right ventricle was grossly undervalued and considered to function mainly as a conduit, while its contractile performance was considered haemodynamically unimportant. Since the early 1950s, however, the relevance of the chamber in the maintenance of normal cardiac physiology was recognized in several cardiovascular disorders.

Tricuspid annular plane systolic excursion (TAPSE) has been proposed as a simple and reproducible parameter for quantitative assessment of RV ejection fraction. It provides a simple method for global RV functional assessment and is a strong predictor of prognosis in heart failure. Although the right ventricle can now be imaged and studied in several ways, two-dimensional (2D) guided M-mode echocardiography is an attractive tool due to its simplicity.

Studies on RV function among patients with hypertensive heart disease (HHD) are rather few and have focused mainly on the diastolic function of the chamber.

Aim of Work
This study is aiming to assess the systolic function of the right ventricle in patients with systemic hypertension using tricuspid annular plane systolic excursion (TAPSE).

Chapter (1)
Systemic Hypertension
• Definition of Hypertension
The cut-off mark for the definition of hypertension has evolved over time. Hypertension is defined as a systolic blood pressure (SBP) of 140 mm Hg or more, or a diastolic blood pressure (DBP) of 90 mm Hg or more, or taking antihypertensive medication.
• Classification of hypertension
Hypertension has been classified according to 2013 ESH/ESC guidelines, as shown in Table 1.
• Prevalence of Hypertension
Hypertension is one of the most significant risk factors for cardiovascular diseases and cerebro-vascular, which ranks as the first and third most frequent causes of death in elderly population all over the world. Blood pressure is a complex genetic trait with heritability estimates of 30–50%, but the intrinsic origin of essential hypertension remains obscure although many environmental factors are known.
Available data on the prevalence of hypertension and the temporal trends of BP values are limited comparable data. Overall the prevalence of hypertension appears to be around 30–45% of the general population, with a steep increase with ageing, also a noticeable difference appear to be in the average BP levels across countries, with no systematic trends towards BP changes in the past decade.
Stroke mortality is a good candidate, because hypertension is by far the most important cause of this event. A close relationship between prevalence of hypertension and mortality for stroke has been reported.
• Path-physiology of hypertension
The path-physiology of hypertension is an area of active research, attempting to explain causes of hypertension, which is a chronic disease characterized by elevation of blood pressure. Hypertension can be classified as either essential or secondary. Essential hypertension indicates that no specific medical cause can be found to explain a patient’s condition. About 90-95% of hypertension is essential hypertension. Secondary hypertension indicates that high blood pressure is a result of another underlying condition, such as kidney disease or tumors (adrenal-adenoma or pheochromocytoma). Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure and arterial aneurysm, and is a leading cause of chronic renal failure.
Most mechanisms leading to secondary hypertension are well understood. The path-physiology of essential
hypertension remains an area of active research, with many theories and different links to many factors. Cardiac and peripheral resistance are the two determinants of arterial pressure. Hence, for understanding the pathogenesis and treatment of hypertensive disorders, it is useful to understand factors involved in the regulation of normal and elevated arterial blood pressure. Cardiac output (CO) and peripheral vascular resistance (PVR) are the two determinants of arterial blood pressure (ABP), ABP = CO X PVR.

Cardiac output is the volume of the blood pumped by the heart in a specific period of time (usually 1 minute), while peripheral vascular resistance is the force in the blood vessels that the left ventricle must overcome to eject blood from the heart. Resistance of blood flow is determined primarily by the diameter of blood vessels and blood viscosity. Increased peripheral vascular resistance results from a narrowing of the arteries and arterioles or and increased in fluid volume in the blood vessels that results from sodium and water retention. Increased peripheral vascular resistance is the most prominent characteristic of hypertension.

The aging process is associated with multiple structural and functional alterations in cardiovascular system that can influence blood pressure regulation. Arterial stiffness, especially in the large arteries, is the pathological characteristic that best exemplifies geriatric hypertension. Histologically, the changes are apparent in the vascular sub endothelial and media layers, which thickness of the arteries due to the accumulation of collagenous fibers, calcium deposition, and loss of elastic fibers, resulting in narrowing and increased stiffness of blood vessels.

It is directly leads to increase in peripheral vascular resistance, a pathogenic characteristic of hypertension in the elderly population. Moreover, with increasing the age, blood vessels also become less responsive to B-adrenergic stimulation, which is necessary for vasodilatation. On the other hand, alpha-adrenergic responsiveness remains unchanged. These changes also contribute to increase peripheral vascular resistance and lead to hypertension.

Beyond this structural change in the arteries, the regulation of vascular resistance is also affected by age related changes in the autonomic nervous system. There is an age associated decline in the sensitivity of the arterial baroreceptor. This effects the regulation of vascular resistance in two important ways. First, a larger change in blood pressure is required to stimulate the baroreceptor to invoke the appropriate compensatory response in heart rate. This also contributes to the age related increase in blood pressure variability. Second, the decrease in baroreceptor sensitivity leads to relatively greater activation of sympathetic nervous system outflow for a given level of blood pressure. An age associated increase in sympathetic nervous system activity has been demonstrated by higher plasma epinephrine and nor epinephrine levels.

These hormones called catecholamine are vasoconstrictors which they cause the blood pressure to constrict making the diameter smaller. By constricting blood vessels, nor-epinephrine increases peripheral vascular resistance and raises blood pressure. Epinephrine constricts blood vessels and increases the force of cardiac contraction, causing blood pressure to rise. Regulation of peripheral vascular resistance by the vascular endothelium is also changed in relation to age. the cell of which become smaller and less uniformly aligned, this change may result in decrease production of endogenous vasodilating substances (e.g., Nitric Oxide) and decline in local control of vascular tone. Impaired nitric oxide – mediated vasodilatation is a potential contributor to the age related increase in peripheral vascular resistance.

Age related changes in renal function, particularly in renal regulation of sodium balance may also contribute to an increase in blood pressure. Decreased renal blood flow and glomelular filtration rate impair the aging kidney’s ability to excrete a sodium load. These renal changes in the regulation of sodium balance create a tendency for sodium retention. This likely plays a part in the finding that a high proportion of older hypertensive individuals, perhaps as high as two thirds, are characterized as having salt sensitivity. Slat sensitivity is operationally defined as an increase in mean arterial blood pressure, commonly 5 mmHg or more, during a high compared to a low dietary sodium intake.

Additionally, aging also alter the Rennin – Angiotensin – Aldosterone – System (RAAS). This affects blood pressure through control of angiotensin II, which has been found to be responsible for sodium and volume retention, vasoconstriction, sympathetic activation, cell growth and proliferation, and possibility atherogenesis. With age, plasma rennin levels decline, and rennin response to sodium depletion, diuretic administration, and upright posture declines as well.

• Risk factors for hypertension
In most cases, the underlying causes of hypertension remain unknown. These risk factors can be classified as modifiable and non-modifiable. Modifiable determinants include factors that can be altered, such as individual and community influences, living and working condition, and socio-cultural factors. On the other hand, non-modifiable determinants include those factors that are beyond the control of the individual.

A- Non-modifiable risk factors:
Non-modifiable risk factors are inherited characteristics in a particular individual that cannot be changed such as age, sex, race or ethnicity, and heredity.

i. Age
Age is the most powerful risk factor for developing hypertension. The worldwide increase in the elderly population (age ≤65 years) is associated with concurrent increases in the prevalence of systemic hypertension and morbidity and mortality from vascular complications of hypertensive disease.
- Cardiovascular disease becomes increasingly common with advancing age. As a person gets older, the heart undergoes subtle physiological changes, even in the absence of disease.
- The heart muscle of the aged heart may relax less completely between beats, and as a result, the pumping chambers become stiffer and may work less efficiently.
- The combination of changes probably reflects stiffening of the blood vessels (reduced arterial compliance) and leads to a large increase in pulse pressure with aging.
- Age-related hypertension appears to be predominantly systolic rather than diastolic. Both systolic blood pressure and diastolic blood pressure increase with age. Systolic blood pressure rises progressively until the age of 70 or 80 years, whereas diastolic blood pressure increases until the age 50 or 60 years and tends to level off or even decline slightly.

ii. Sex
The overall incidence of hypertension is higher in men than in women until the age 55 years. After that, women’s risk for hypertension increases sharply. The National Health and Nutrition Examination Survey (NHANES) in USA (2003) showed that, the prevalence of hypertension was higher in women than in men aged 70 years old and above.

iii. Heredity
Family history of hypertension among first degree relatives (e.g. parents, siblings, and off-spring) is considered a risk factor for developing hypertension. First degree relatives of patients with hypertension have two-fold greater risk of hypertension, and the risk increases to four-fold when more family member are hypertensive. A family history of hypertension at an early age increases the risk.

iv. Race or Ethnicity
Prevalence of hypertension is twice as high in African American as in whites. The reason for the increased prevalence of hypertension among blacks is unclear, but the increase has been attributed to lower rennin levels, greater sensitivity to vasopressin, higher salt intake, and greater environmental stress.

B. Modifiable risk factors:
Modifiable risk factors could be controlled through life style modification or by medical intervention. These risk factors include diabetes mellitus, elevated serum lipids, sleep apnea, and unhealthy lifestyle as physical inactivity, consumption of unhealthy diet, obesity, smoking, excessive alcohol or caffeine intake, and poor stress management.

i. Diabetes mellitus
Patients with diabetes have 1.5 to 2 time increased risk of having hypertension. WHO reported that, about 60 to 65% of people with diabetes have high blood pressure.
Diabetes mellitus accelerates atherosclerosis via numerous metabolic events: chronic hyperglycemia, insulin resistance, and dyslipidemia, which alter the function of multiple cell types, including endothelium, smooth muscle cells, and platelets leading to build up of atheroma in the arterial wall.
Insulin resistance is a cardinal defect in type II diabetes in elderly. Insulin resistance and hyper-insulinemia are associated with hypertension. The mechanism of developing hypertension in elderly with insulin resistance may be due to increase the activity of sympathetic nervous system leading to increases in cardiac output and peripheral vascular resistance.
Recent evidence suggests that glycemic control may decrease the hypertension risk. Keeping sugar well controlled minimize potential damage to blood vessels, offering strong protection against the development
of high blood pressure.

ii. Elevated serum lipids

Raised blood cholesterol increases the risk of heart disease and stroke. Elevated levels of serum lipids (cholesterol and triglycerides) are extremely common and are one of the most important risk factors that can be changed. Lowering raised blood cholesterol reduces the risk of heart disease. The prevalence of raised total cholesterol noticeably increases according to the income level of the country. In low-income countries, around 25 percent of adults have raised total cholesterol, while in high-income countries; over 50 percent of adults have raised total cholesterol. The major lipid particles, cholesterol and triglycerides, both have important functions in the body. Cholesterol is an essential component of the cell membranes functioning to provide stability while permitting membrane transport. It is a precursor to adrenal steroids, sex hormones, bile and bile acids. Triglycerides are the major source of energy for the body. Both cholesterol and triglycerides are in soluble molecules and are transported in the circulation as lipoproteins. Normal serum cholesterol level is below 200mg/dl. When there is too much cholesterol in the body because of diet and the rate at which the cholesterol is processed, it is slowly build up in the inner lining of the arteries. This can lead to narrowing of the arteries and build up atherosclerosis.

Most cholesterol in the blood is carried in two kinds of a protein called (lipoproteins): low-density lipoprotein (LDL), and high density lipoprotein (HDL). Low-density lipoprotein cholesterol, which is “bad” cholesterol causing the build-up of plaque. The optimal level of LDL cholesterol is less than 100 mg/dl. High-density lipoprotein cholesterol is the “good” cholesterol because it tends to carry excess cholesterol back to the liver where it is removed from the body. The HDL level of 60 mg/dl and above is considered protective against cardiovascular disease (CVD). People with a low level of HDL cholesterol (less than 40 mg/dl) have a higher risk of cardiovascular disease.

Triglycerides are the most common type of fat in the body. The normal triglycerides level is less than 150 mg/dl. Many people who have CVD or DM have high triglycerides level combined with low HDL cholesterol or high LDL cholesterol seems to speed up atherosclerosis. Epidemiological study conducted in Japan (2004) has shown that, the level of total cholesterol in the blood is a strong predictor of elevated blood pressure. Other studies confirmed that the relation between the incidence of hypertension and high plasma cholesterol levels is gradually attenuated after the age of 65 years. Decreasing fat content in the diet is the first step in reducing cholesterol levels. Several clinical trials have shown the efficiency of lipid lowering agent as statins for reducing cardiovascular risk in patients with elevated serum lipid.

iii. Sleep apnea

Obstructive sleep apnea (OSA) is the most common category of sleep-disordered breathing. The term “sleep-disordered breathing” is commonly used in the U.S. to describe the full range of breathing problems during sleep in which not enough air reaches the lungs (hypopnea and apnea).

Hypertension occurs in more than 50% of individuals with obstructive sleep apnea. Approximately 70% of patients with obstructive sleep apnea are obese. Hypertension related to (OSA) should also be considered in patients with drug–resistant hypertension and in patients with a history of snoring. Despite extensive research, the underlying mechanisms of obstructive sleep apnea induced hypertension are not entirely understood. Evidence indicates that sympathetic activation plays a central role on OSA–induced hypertension.

iv. Lifestyle factors

Lifestyle changes are important for preventing and treating high blood pressure. Healthy changes include maintaining a normal weight, exercising regularly, quitting smoking, limiting alcohol consumption to no more than one or two drinks a day, reducing sodium (salt) intake, and increasing potassium intake.

• Lifestyle factors include:
  A) Physical inactivity:
  Lack of physical activity increases the risk of blood vessel disease, heart disease, and stroke. It also makes it easier to put on unwanted pounds. In addition, when you are out of shape, it takes more effort for your heart to pump blood. This increases the force exerted on arteries, which can lead to high blood pressure. Regular exercise and increasing amount of physical activity have been shown to favorably affect positively on blood pressure in people with hypertension, independent of changes in body weight and also prevent hypertension.
in normo-tensive individuals. An epidemiological study in USA illustrates that regular exercise and physical activity can attenuate systolic and diastolic blood pressure by 11 and 8 mmHg respectively.

B) Unhealthy diet:
A diet that’s high in calories, fats and sugars and low in essential nutrients contributes directly to poor health as well as to obesity. In addition, there are some problems that can happen from eating too much salt. Some people are "salt sensitive," meaning a high-salt (sodium) diet raises their high blood pressure. Salt keeps excess fluid in the body that can add to the burden on the heart. While too much salt can be dangerous, healthy food choices can actually lower blood pressure. High dietary sodium intake is associated with an increased incidence of hypertension. At least 40% of elders who eventually develop hypertension are salt sensitive and the excess salt may be the precipitating cause of hypertension in these individuals. Decrease intake of fresh fruits and vegetables has been proposed to be a vascular risk factor. Most of this food are antioxidants and protect the arterial intima from oxidative damage. Diet low in potassium is associated with an increased risk of hypertension. Some clinical trials suggest that increasing dietary potassium by approximately (54 mmol) per day can reduce systolic blood pressure by 2-3 mmHg in hypertensive patients.

C) Obesity:
The prevalence of obesity is increasing drastically in the recent decade and it has become particularly high in the elderly population. The prevalence of overweight and obesity is commonly assessed by using body mass index (BMI), defined as the weight in kilograms divided by the square of the height in meters (kg/m2). A BMI ≤ 25 kg/m2 is defined as overweight and a BMI ≤ 30 kg/m2 as obese.
In the elderly, obesity has been associated not only with increased mortality, but also with elevated risks of hypertension and other CVD. Anyone who is overweight has a risk for developing hypertension 50% more than people with normal weight.

D) Smoking:
Cigarette smoking is a major risk factor for high blood pressure. Cigarette smokers have twice the chance of developing hypertension and four times the chance of sudden death compared with non smokers. Smoking is one of the leading causes of preventable death in the United States, and it increases the risk of having a heart attack or stroke. With each cigarette you smoke, blood pressure also shoots up by as much as 10 points and stays higher for up to an hour. If you constantly have a cigarette in your hand, that could keep your blood pressure elevated for much of the day. High amounts of secondhand smoke, environmental smoke, and passive smoking can also contribute to high blood pressure; it is best to avoid cigarette smoke as much as possible. The mechanism of hypertension risk associated with smoking is complex. The main mechanism appears to be through the development and progression of atherosclerosis.
Cigarette tobacco contains high concentration of oxidants and free radicals (like Nitric oxide), as well as nicotine, a major constituent of cigarettes. These constituents are considered to be absorbed into the systemic circulation, to injure the arterial endothelium and thus promote atherogenesis. Nicotine has been suggested to contribute to atherosclerosis via its effect on changes in the lipid metabolism, endothelial damage and production of growth factors. Smoking also increases insulin resistance enhances sympathetic activity which increases heart rate and blood pressure.

E) Alcohol consumption:
Heavy and regular use of alcohol can increase blood pressure dramatically. It can also cause heart failure, lead to stroke and produce irregular heartbeats. Too much alcohol can contribute to high triglycerides, cancer and other diseases, obesity, alcoholism, suicide and accidents. The effect of alcohol appears to be depending on the amount consumed. Reducing alcohol gradually in hypertensive patients can lower blood pressure by average of 13 mmHg systole and 15 mmHg diastole. However, the abrupt cessation of alcohol intake in individuals consuming great amounts resulted in a rapid increase in their blood pressure.

F) Caffeine consumption:
Caffeine can cause a short-lived but dramatic rise in blood pressure. The amount of caffeine in two to three cups of coffee can raise systolic pressure 3 to 14 millimeters of mercury (mm Hg). Diastolic pressure can be increased 4 to 13 mm Hg. However, this transient rise in blood pressure due to caffeine has not been shown to increase risk of hypertension. Caffeine actually increases blood pressure, but the association between habitual consumption of caffeinated beverages and incidence of hypertension is uncertain. However, a recent
A longitudinal study found a positive association between caffeine consumption and blood pressure.

G) Poor stress management:
Severe stress can lead to a temporary but dramatic spike in blood pressure. Over time, this might contribute to high blood pressure, although that has never been conclusively proved. In addition, some people cope with stress by overeating, drinking too much, or smoking. A study carried out in Brazil shows that people exposed to high levels of repeated psychological stress develop hypertension to a greater extent than those who do not experience as much stress.

Left Ventricular Hypertrophy in Hypertension

Left ventricular hypertrophy (LVH) is defined as an increase in the mass of the left ventricle, which can be secondary to an increase in wall thickness, an increase in cavity size, or both. LVH as a consequence of hypertension usually presents with an increase in wall thickness, with or without an increase in cavity size. This increase in mass predominantly results from a chronic increase in afterload of the LV caused by the hypertension, although there is also a genetic component. A significant increase in the number and/or size of sarcomeres within each myocardial cell is the pathologic mechanism.

Left ventricular wall thickness and/or mass is the best studied marker of hypertensive heart disease. Increased left ventricular wall thickness and mass have continuously been found to be associated with the level of blood pressure and age. However, without increased systolic blood pressure, clinically significant increases in left ventricular mass do not occur with advancing age. Chronic systolic hypertension therefore seems to be the principal cause of left ventricular hypertrophy.

LVH serves as an integrated surrogate for cumulated blood pressure load and is best described as being proportional to the area under the lifetime BP curve. This is also supported by the fact that there is a strong association of left ventricular mass and mean 24-hour ambulatory blood pressure. The increase of LV mass with age might reflect the influence that other risk factors exert with time on the development of LVH. The relationship between echocardiographic LV mass and clinical blood pressure is usually weak. Twenty-four-hour blood pressure recordings have shown a much closer correlation between LV mass and average daily blood pressure. Non-haemodynamic factors, such as age, sex, race, body mass index, diabetes, and dietary salt intake, may contribute to determine who among hypertensive patients develop LVH and to what degree LVM is increased.

In fact, the coexistence of hypertension with diabetes increases the prevalence of LVH. Moreover, insulin resistance and high insulin levels are associated with the development of LVH in hypertensive patients. Other major cardio-metabolic risk factors, notably hypercholesterolaemia and hyperglycaemia, may also modify the extent of LVM and the prevalence of LVH in the hypertensive population. Genetic factors might also exert a powerful modulation of LV mass; in fact monozygotic twins have more similar LV mass values than dizygotic twins.

• Pathogenesis of Left Ventricular Hypertrophy:
Two major triggers for LVH are biomechanical stress and neurohormonal factors. LVH is mainly due to pressure or volume overload on the heart. Common causes of pressure overload are systemic hypertension, aortic stenosis, coarctation of the aorta and hypertrophic cardiomyopathy. It is thought that a mechanical signal initiates a cascade of biological events which lead to coordinated cardiac growth. There is then an increased myosin heavy chain synthesis (by about 35%) within hours of pressure overload. This increase is initially predicted by an increase in translational efficiency.

Neurohormonal factors that have been implicated in left ventricular hypertrophy include: Angiotensin II, endothelin, calcineurin, metalloproteinases and heterometrimeric G.

Angiotensin II
It has been postulated that angiotensin II, via the AT1 receptor, plays a key role in the induction of hypertrophy because it can directly induce the molecular events of early cardiac growth. Cardiac rennin-angiotensin system has been proposed as an important determinant of hypertrophic response. The importance of angiotensin II in the development of LVH in hypertensive subjects is also suggested indirectly by the observation that an ACE inhibitor causes regression of left ventricular hypertrophy, more than other anti-hypertensive drugs.

Endothelin
– Some animal studies suggest that endothelin plays a role in the development of left ventricular wall hypertrophy in response to elevated blood pressure.

Calcineurin
– Calcineurin is a calcium calmodulin–dependent phosphatase. It serves as a master switch for clinical hypertrophy. In animal studies, that transgenic mice that over-express components of the calcineurin signaling pathway, develop a hypertrophic phenotype that can be expressed by pharmacological inhibitors of calcineurin.

Metalloproteinase (MMPs)
– Matrix metalloproteinase is a family of zinc dependant interstitial enzymes. Their tissue inhibitors (TIMPs) control the breakdown of collagen. The role of MMPs in concentric hypertrophy is not fully understood, but preliminary observations show that they are activated in experimental pressure overload hypertrophy. Studies have also shown that imbalance between MMPs and TIMPs could lead to LVH and diastolic dysfunction.

Heterometrimeric G Proteins
– Many hormones and neurotransmitters implicated in the initiation and exacerbation of myocardial hypertrophy including angiotensin II and endothelin, bind to cell membrane receptors which couple to a subset of intracellular hetero-metrimeric G proteins – the G (q) class.

About Essay Sauce

EssaySauce.com is a completely free resource to help students research their academic work and learn from great essays!

View all posts by Essay Sauce

...(download the rest of the essay above)
Latest reviews:

- Health essays
- Tourism
- Public Relations and Crisis Management in Tertiary Institutions

Search for student essays:

Search ...

About EssaySauce, the student essay site:

EssaySauce.com is a free resource for students, providing thousands of example essays to help them complete their college and university coursework. Students can use our free essays as examples to write their own.
Latest student essays:

- Ocular disease
- HUMAN action recognition
- Analysing data production
- Desorption study
- Surfactants (surface active agents)
- Islamic Finance and Its Impact on Customer Satisfaction
- Persian gulf
- Feminist approach (Bhumika) (notes)
- What does it mean to be a Muslim woman in 21st century? (Shari’ah)
- Appellate Body’s analysis under section XIV(c)

Student essay categories:

- Accounting essays
- Architecture essays
- Business essays
Average review:

Overall rating: 0 out of 5 based on 0 reviews.
Q: Is EssaySauce.com free?

Yes! EssaySauce.com is a completely free resource for students. You can view our terms of use here.

Why use Essay Sauce?

The brightest students know that the best way to learn is by example! EssaySauce.com has thousands of great essay examples for students to use as inspiration when writing their own essays.

Is Essay Sauce completely free?

Yes! EssaySauce.com is a completely free resource for students. You can view our terms of use here.

Info:

About
Content policy
Essay removal request
Privacy
Terms of use