Excellence

Medical Beauty Training

B12 Injection Manual

Contents

What is Vitamin B12

Vitamin B12, also called cobalamin, is a water-soluble vitamin that is involved in the metabolism of every cell of the human body: it is a cofactor in DNA synthesis, and in both fatty acid and amino acid metabolism. It is particularly important in the normal functioning of the nervous system via its role in the synthesis of myelin and in the maturation of developing red blood cells in the bone marrow.

Vitamin B is one of eight vitamins; it is the largest and most structurally complicated vitamin. It consists of a class of chemically related compounds (vitamers), all of which show physiological activity. It contains the biochemically rare element cobalt (chemical symbol Co) positioned in the centre of a corrin ring. The only organisms to produce vitamin B12 are certain bacteria, and archaea. Some of these bacteria are found in the

soil around the grasses that ruminants eat; they are taken into the animal, proliferate, form part of their gut flora, continue to produce vitamin B12.

There are no naturally occurring notable vegetable dietary sources of the vitamin, so vegans and vegetarians are advised to take a supplement or fortified foods. Otherwise, most omnivores people in developed countries obtain enough vitamin B12 from consuming animal products, including meat, milk, eggs and fish. Staple foods, especially those that form part of a vegan diet, are often fortified by having the vitamin added to them.

Vitamin B12 supplements are available in single agent or multivitamin tablets; and pharmaceutical preparations may be given by intramuscular injection. The most common cause of vitamin B12 deficiency in developed countries is impaired absorption due to a loss of gastric intrinsic factor, which must be bound to food-source B12 in order for absorption to occur. Another group affected are those on long term antacid therapy,
using proton pump inhibitors. H2 blockers or other antacids. The condition may be characterised by limp neuropathy or a blood disorder called pernicious anemia, a type of megaloblastic anemia. Folate levels in the individual may affect the course of pathological changes and symptomatology. Deficiency is more likely after the age of 60, and increases in incidence with advancing age. Dietary deficiency is very rare in developed countries due to access to dietary meat and fortified foods, but children in some regions of developing countries are at particular risk due to increased requirements during growth coupled with lack of access to dietary B12; adults in these regions are also at risk. Other causes of vitamin B12 deficiency are much less frequent.

Methylcobalamin

Methylcobalamin is a form of vitamin B12. Physically it resembles the other forms of Vitamin B12, occurring as dark red crystals that freely form cherry-coloured transparent solutions in water. B12 is the most chemically complex of all the vitamins. The structure of B12 is based on a corrin ring, which is similar to the porphyrin ring found in haem. The central metal ion is cobalt. Four of the six coordination sites are provided by the corin ring, and a fifth by being a cyano group (-CN), a hydroxyl group (-OH), a methyl group (-CH3) or a 5'-deoxyadenosyl group (here the C5' atom of the deoxyribose forms the covalent bond with cobalt respectively, to yield the four vitamers (forms) of B12. Historically, the covalent C-Co bond is one of the first examples of carbon-metal bonds to be discovered in biology. The hydrogenases and, by necessity, enzymes associated with cobalt utilization, involve metal-carbon bonds. Vitamin B12 is a generic descriptor name referring to a collection of cobalt and corrin ring molecules which are defined by their particular vitamin function in the body. All of the substrate cobalt-corrin molecules from which B12 is made must be synthesized by bacteria.After this synthesis is complete, the human body has the ability (except in rare cases) to convert any form of B12 to an active form, by means of enzymatically removing certain prosthetic groups from the cobalt atom and replacing them with others.

Vitamers

The four vitamers of B12 are all deeply red-coloured crystals and water solutions, due to the colour of the cobalt-corrin complex.

Cyanocobalamin is one form of B12 because it can be metabolized in the body to an active coenzyme form. The cyanocobalamin form of B12 does not occur in nature normally, but is a by-product of the fact that other forms of B12 are avid binders of cyanide (-CN) which they pick up in the process of activated charcoal purification of the vitamin after it is made by bacteria in the commercial process. Since the cyanocobalamin form of B12 is easy to crystalize and is not sensitive to air-oxidation, it is typically used as a form of B12 for food additives and in many common multivitamins. Pure cyanocobalamin possesses the deep pink colour associated with most octahedral cobalt (ll) complexes and the crystals are well formed an easily grown up to millimetre size.

Hydroxocobalamin is another vitamer of B12 commonly encountered in pharmacology, but is normally present in the human body.

Hydroxocobalamin is sometimes denoted

B12a. This is the form of B12 produced by bacteria, and which is converted to cyanocobalamin in the commercial charcoal filtration step of production. Hydroxocobalamin has an avid affinity for cyanide ions and has been used as an antidote to cyanide poisoning. It is supplied typically in water solution for injection.

Hydroxocobalamin is thought to be converted to the active enzymatic forms of B12 more easily than cyanocobalamin, and since it is a little more expensive than cyanocobalamin, and has longer retention times in the body, has been used for vitamin replacement in situations where added reassurance of activity is desired. Intramuscular administration of hydroxocobalamin is also the preferred treatment for pediatric patients with intrinsic cobalamin metabolic diseases, for vitamin B12 deficient patients with tobacco amblyopia (which is thought to perhaps have a component of cyanide poisoning from cyanide in cigarette smoke); and for treatment of patients with pernicious anemia who have optic neuropathy. Adenosylcobalamin (adoB12) and methylcobalamin (MeB12) are the two enzymatically active cofactor forms of B12 that naturally occur in the body. Most of the body's reserves are stored as adoB12 in the liver.

These are converted to the other methylcobalamin form as needed.

Dietary Requirements

The U.S Institute of Medicine (now known as the National Academy of Medicine since 2015) updated Estimated Average Requirements (EARs) and Recommended Dietary Requirements (RDAs) for Vitamin B12 in 1998. The EAR for vitamin B12 for women and men ages 14 and up is 2.0 ug/day; the RDA is 2.4 ug/day.RDAs are higher than EARs so as to identify amounts that will cover people with higher than average requirements. RDA for pregnancy equals 2.6 ug/day. RDA for lactation equals 2.8 ug/day. For infants up to 12 months the Adequate intake (AI) is 0.4-0.5ug/day. (ALs are established when there is insufficient information to determine EARs and RDAs.) For children ages 1-13 years the RDA increases with age from 0.9 to 1.8ug/day. Because 10 to 30 percent of older people may be unable to effectively absorb vitamin B12 naturally occurring in foods, it is advisable for those older than 50 years to meet their RDA mainly by consuming foods fortified with vitamin B12 or a supplement containing vitamin B12. As for safety, tolerable Upper Intake Levels (known as ULs) are set for vitamins and minerals when evidence is sufficient. In the case of vitamin B12 there is no UL, as there is no human data for adverse effects from high doses. Collectively the EARs,RDAs AIs and ULs are referred to as dietary reference intakes (DRIs)

The European Food Safety Authority (EFSA) refers to the collective set of information as Dietary Reference Values, with Proportion Reference Intake (PRI) instead of RDA, and Average Requirement instead of EAR. AI and UL defined the same in the United States.For women and men over the age of 18, the adequate intake (AI) is set at 4.0 ug/day. AI for pregnancy is 4.5ug/day, for lactation 5.0ug/day. For children aged 1-17 years the AIs increase with age from 1.5 ug/day to 3.5 ug/day. These AIs are higher than the U.S RDAs. The EFSA also reviewed the safety question and reached the same conclusion as in the United States - that there was not sufficient evidence to set a UL for vitamin B12. For U.S food and dietary supplement labelling purposes the amount in a serving is expressed as a percentage of Daily Value (%DV). For vitamin B12 labelling purposes 100% of the daily value was 6.0 ug, but as of May 27, 2016 was revised downward to 2.4 ug. A table of the old and new adult Daily Values is provided at Reference Daily Intake. The original deadline to be in compliance was July 28, 2018, but on September 29, 2017
the FDA released a proposed rule that extended the deadline to January 1, 2020 for large companies and January 1, 2021 for small companies.

Sources

Most omnivorous people in developed countries obtain enough vitamin B12 from consuming animal products, including meat, fish, eggs and milk, but there are no vegan sources other than B12-fortified foods or B12 supplements.

Bacteria and Archaea

B12 is only produced in nature by certain bacteria, and achaea. It is synthesized by some bacteria in the gut flora in humans and other animals, but humans cannot absorb this as it is made in the colon, downstream from the small intestine, where the absorption of most nutrients occurs.
Ruminants, such as cows and sheep, absorb B12 produced by bacteria in their guts. For gut bacteria of ruminants o produce B12 the animal must consume sufficient amounts of cobalt. These grazing animals acquire the bacteria that produce vitamin B12, and the vitamin itself.
Faeces are a rich source of vitamin B12, and are eaten by many animals, including dogs and cats. Lagomorpha species, including rabbits and hares, form faecal pellets in their ceacum called cecotropes, which consists of chewed plant material that has been metabolized by cecal bacteria; cecotropes contain digestible carbohydrates and B vitamins synthesized by the resident bacteria. These animals ingest cecotropes which have been expelled in their faeces.

Animals

Animals store vitamin B12 in liver and muscle and some pass the vitamin into their eggs and milk;meat, liver, eggs and milk are therefore sources of the vitamin for other animals as well as humans. For humans, the bioavailability from eggs is less than 9% compared to 40% to 60% from fish, fowl and meat. Insects are a source of B12 for animals (including other insects and humans).
Food sources with a high concentration of vitamin B12-50 to 99 ug B12 per 100 grams of food-include clams; liver and other organ meats from lamb, veal, beef and turkey;mackerel; and crab meat.

Plants and Algae

Natural sources of B12 include dried and fermented plant foods, such as tempeh nori and laver, seaweed. Many other types of algae are rich in vitamin B12, with some species, such as Porphyra yezoensis, contains as much cobalamin in liver.

Fortified foods

The U.K vegan society, the vegetarian Resource Group, and the Physicians Committee for Responsible Medicine, among others, recommend that every vegan who is not consuming adequate B12 from fortified foods take supplements. Foods for which B12-fortified versions are widely available include breakfast cereals, soy products, energy bars, and nutritional yeast.

Supplements

Vitamin B12 is included in multivitamin pills; and in some countries grain-based foods such as bread and pasta are fortified with B12. In the U.S non-prescription products can be purchased proving up to 5'000 ug per serving, and it is a common ingredient in energy drinks and energy shots, usually at many times the recommended dietary allowance of B12. The vitamin can also be a prescription product via injection or other means. Tablets have sufficiently large quantities of the vitamin such that 1% to 5% of the free crystalline B12 is absorbed along the entire intestine by passive diffusion.

Sublingual methylcobalamin, which contains no cyanide, is available in 5-mg tablets. The metabolic rate and biological distribution of methylcobalamin are expected to be similar to that of other sources of vitamin B12 in the diet, but the amount of cyanide in cyanocobalamin even in the largest available dose-20 ug of cyanide in a 1'000-ug cyanocobalamin tablet-is less than the daily consumption of cyanide from food, and so cyanocobalamin is not considered a health risk.

Parental administration

Injection and patches are sometimes used if digestive absorption is impaired, but this course of action may not be necessary with high-potency oral supplements (such as 0.5-1 mg or more). Even pernicious anemia can be treated entirely by the oral route. If the person has inborn errors in the methyl transfer pathway (cobalamin C disease, combined methylmalonic aciduria and homocystinuria), treatment with intravenous, intramuscular hydroxocobalamin or transdermal B12 is needed.

Pseudovitamin-B12 Pseudovitamin-B12 refers to B12-like analogues that are biologically inactive in humans and yet found to be present alongside B12 in humans, many food sources (including animals), and possibly supplements and fortified foods. Most cyanobacteria, including Spirulina, and some algae, such as dried Asakusa-nori (Porphyra tenera), have been found to contain mostly pseudovitamin-B12 instead of biologically active B12. In one common form of pseudo B12 available to Salmonella enterica serovar Typhimurium,

Biochemistry

Enzyme Function

If folate is present in quantity, then of the two absolutely vitamin B12-dependant enzyme family reactions in humans, the MUT-family reactions show the most direct and characteristic secondary effects, focusing on the nervous system (see below). This is because the MTR (methyltransferase-type) reactions are involved in regenerating folate, and thus are less evident when folate is in good supply. Since the late 1990s, folic acid has begun to be added to fortify flour in many countries, so folate deficiency is now rarer. At the same time, since DNA synthetic-sensitive tests for anemia and erythrocyte size are routinely done in even simple medical test clinics (so that there's folate-mediated biochemical effects are more often directly detected), the MTR dependant effects of B12 deficiency are becoming apparent not as anemia due to DNS synthetic problems (as they were classically), but now mainly as a simple and less obvious elevation of homocysteine in the blood and urine (homocystinuria). This condition may result in long term damage to arteries and in clotting (stroke and heart attack), but this effect is difficult to separate from other common processes associated with atherosclerosis and aging. The specific myelin damage resulting from B12 deficiency, even in the presence of adequate folate and methionine, is more specifically and clearly a vitamin deficiency problem. It has been connected to B12 most directly by reactions related to MUT, which is absolutely required to convert methylmalonyl coenzyme A into succinyl coenzyme A. Failure of this second reaction to occur results in elevated levels of MMA, a myelin destabilizer. Excessive MMA will prevent normal fatty acid synthesis, or it will be incorporated into fatty acid itself rather than normal malonic acid. If this abnormal fatty acid subsequently is incorporated into myelin, the resulting myelin will be too fragile and demyelination will occur. Although the precise mechanism or mechanisms are not known with certainty, the result is subacute combined degeneration of spinal cord. Whatever the cause, it is known that B12 deficiency causes neuropathies, even if folic acid is present in good supply and therefore anaemia is notpresent.

Vitamin B12-dependant MTR reactions may have neurological effects through an indirect mechanism. Adequate methionine (which, like folate, must otherwise be obtained in the diet, if it is not regenerated from homocysteine by a B12 dependant reaction) is needed to make S-adenosylmethionine (SAMe), which in turn necessary for methylation of myelin sheath phospholipids. Although production of SAMe is not B12 dependent, help in recycling for provision of one adequate substrate for it (the essential amino acid methionine) is assisted by B12. In addition, SAMe is involved in the manufacture of certain neurotransmitters, catecholamines and in brain metabolism.

These neurotransmitters are important for maintaining mood, possibly explaining why depression is associated with B12 deficiency. Methylation of the myelin sheath phospholipids may also depend on adequate folate, which in turn is dependent on MTR recycling, unless ingested in relatively high amounts.

Physiology

Absorption

Methyl-B12 is absorbed by two processes. The first is an intestinal mechanism using intrinsic factor through which 1-2 microorganisms can be absorbed every few hours. The second is a diffusion process by which approximately 1% of the remainder is absorbed.

The human physiology of vitamin B12 is complex and therefore is prone to mishaps leading to vitamin B12 deficiency. Protein-bound vitamin B12 must be released from the proteins by the action of digestive proteases in both the stomach and small intestine.

Gastric acid releases the vitamin from food particles; therefore antacid and acid-blocking medications (especially proton-pump inhibitors) may inhibit absorption of B12. B12 taken in a low-solubility, non-chewable supplement pill form may bypass the mouth and stomach and not mix with gastric acids, but acids are not necessary for the absorption of free B12 not bound to protein; acid is necessary only to recover naturally-occurring vitamin B12 from foods. R-protein (also known as haptocorrin and cobalophilin) ia a B12 binding protein that is produced in the salivary glands. It must wait to bind food-B12 until B12 has been freed from proteins in food by pepsin in the stomach. B12 then binds to the R-protein to avoid degradation of it in the acidic environment of the stomach. This pattern of B12 transfer to a special binding protein secreted in a previous digestive step, is repeated once more before absorption.

The next binding protein for B12 is
intrinsic factor (IF), a protein synthesized by gastric parietal cells that is secreted in response to histamine, gastrin and pentagastrin, as well as the presence of food. In the duodenum, proteases digest R-proteins and release their bound B12, which then binds to IF, to form a complex (IF/B!"). B12 must be attached to IF for it to be efficiently absorbed, as receptors on the enterocytes in the terminal ileum of the small bowel only recognise the B12-OR complex; in addition, intrinsic factor protects the vitamin from catabolism by intestinal bacteria. Absorption of food vitamin B12 thus requires an intact and functioning stomach, exocrine pancreas, intrinsic factor, and small bowl. Problems with any one of these organs makes a vitamin B12 deficiency possible. Individuals who lack intrinsic factor
have a decreased ability to absorb B12. In pernicious anaemia, there is a lack of IF due to autoimmune atrophic gastritis, in which antibodies form against parietal cells. Antibodies may alternately form against and binds to IF, inhibiting it from carrying out its B12 protective function. Due to the complexity of B12 absorption, geriatric patients, many of whom are hyperacidic due to reduced parietal cell function, have an increased risk of B12 deficiency. This results in 80-100% excretion of oral doses in the faeces versus 30-60% excretion in feces as seen in individuals with adequate IF. Once the IF/B12 complex is recognised by specialized ileal receptors, it is transported into the portal circulation. The vitamin is then transferred to transcobalamin II (TC-II/B12), which serves as the plasma transporter. Hereditary defects in production of the transcoal balmains and their receptors may produce functional deficiencies in B12 and infantile megaloblastic aneamia and abnormal B12 related biochemistry, even in some cases with normal blood B12 levels. For the vitamin to serve inside cells, the TC-II/B12 complex must bind to a cell receptor and be endocytosed.

The transcobalamin-II is degraded within a lysosome, and free B12 is finally released into the cytoplasm, where it may be transformed into the proper coenzyme, by certain cellular enzymes (see above). Investigations into the intestinal absorption of B12 point out that the upper limit of absorption per single dose, under normal conditions, is about 1.5 ug. “ studies in normal persons indicated that about 1.5 ug is assimilated when a single dose varying from 5-50 ug is administered by mouth. In a similar study Swendseid et al. stated that the average maximum absorption was 1.6 ug. The bulk diffusion process of B12 absorption noted in the first paragraph above, may overwhelm the complx R-factor and IGF-factor dependant absorption, when oral doses of B12 are very large (a thousand or more ug per dose) as commonly happens in dedicated-pill oral B12 supplementation. It is this last fact which allows pernicious anemia and certain other defects in B12 absorption to be treated with oral megadoses of B12, even without any correction of the underlying absorption defects. See the section on supplements above.

Storage and excretion

The total amount of vitamin B12 stored in the body is about 2-5mg in adults. Around 50% of this is stored in the liver. Approximately 0.1% of this is lost per day by secretions into the gut, as not all these secretions are reabsorbed. Bile is the main form of B12 excretion; most of the B12 secreted in the bile is recycled via enterohepatic circulation. Excess B12 beyond the blood’s binding capacity is typically excreted in urine. Owing to the extremely efficient enterohepatic circulation of B12 the Liver can store 3 to 5 years’ worth of vitamin B12; therefore, nutritional deficiency of this vitamin is rare. How fast B12 levels change depends on the balance between how much B12 is obtained from the diet, how much is secreted and how much is absorbed. B12 deficiency may arise in a year if initial stores are low and genetic factors favourable, or may not appear for decades. In infants, B12 deficiency can appear much more quickly.

Deficiency

Vitamin B12 deficiency

Vitamin B12 deficiency can potentially cause severe and irreversible damage, especially to the brain and nervous system. At levels only slightly lower than normal, a range of symptoms such as fatigue, lethargy, difficulty walking (staggering balance problems) depression, poor memory, breathlessness, headaches and pale skin, among others, may be experienced, especially in elderly people (over age 60) who produce less stomach acid as they age, thereby increasing their probability of B12 deficiency. Vitamin B12 deficiency can also cause symptoms of mania and psychosis. Vitamin B12 deficiency is most commonly caused by low intakes, but can also result from malabsorption, certain intestinal disorders, low presence of binding proteins, and use of certain medications. Vitamin B12 is rare from plant sources, so vegetarians are more likely to suffer from vitamin B12 deficiency. Infants are at higher risk of vitamin B12 deficiency if they were born to vegetarian mothers. The elderly who have diets with limited meat or animal products are vulnerable populations as well. Vitamin B12 deficiency may occur in between 40 to 80% of the vegetarian population who are not also consuming a vitamin B12 supplement. In Hong Kong and India, vitamin B12 deficiency has been found roughly 80% of the vegan population as well. Vegans can avoid this by eating B12 fortified foods like cereals, plant-based milks, and nutritional yeast as a regular part of their diet. In addition to worries concerning those following a vegetarian or vegan diet, research has found that approximately 39% of the general population may have possible B12 deficiencies or difficulty with the absorption of this nutrient. Taking a B12 supplement could be beneficial to most people. B12 is a co-substrate of various cell reactions involved in methylation synthesis of nucleic acids and neurotransmitters. Synthesis of the tri monoamine neurotransmitters can enhance the effects of a traditional antidepressant. The intracellular concentrations of vitamin B12 can be inferred through the total plasma concentration of homocysteine, which can be converted to methionine through an enzymatic reaction that uses 5-methyltetrahydrofolate as the methyl donor group. Consequently, the plasma concentration of homocysteine falls as the intracellular concentration of vitamin B12 rises. The active metabolite of vitamin B12 is required for the methylation of homocysteine in the production of methionine, which is involved in a number of biochemical processes including the monoamine neurotransmitters metabolism. Thus, a deficiency in vitamin B12 may impact the production and function of those neurotransmitters.

Medical uses

Repletion of deficiency

Severe vitamin B12 deficiency is corrected with frequent intramuscular injections of large doses of the vitamin, followed by maintenance doses at longer intervals. Tablets are sometimes used for repletion in mild deficiency; and for maintenance regardless of severity. Vitamin B12 supplementation sometimes leads to acneiform eruptions (acne-like rashes).

Drug interactions

H2-receptor antagonists and proton-pump inhibitors. Gastric acid is needed to release vitamin B12 from protein for absorption. Reduce secretion of gastric acid and pepsin produced by H2blocker or proton-pump inhibitor (PPI) drugs can reduce absorption of protein-bound (dietary) vitamin B12 , although not of supplemental vitamin B12 . H2-receptor antagonist examples include cimetidine, famotidine, nizatidine, and ranitidine. PPIs examples include omeprazole, lansoprazole, rabeprazole, pantoprazole, and esomeprazole. Clinically significant vitamin B12 deficiency and megaloblastic anemia are unlikely, unless these drug therapies are prolonged for two or more years, or if in addition the person's diet is below recommended intakes. Symptomatic vitamin deficiency is more likely if the person is rendered achlorhydric (complete absence of gastric acid secretion), which occurs more frequently with proton pump inhibitors than H2 blockers.

Metformin

Reduced serum levels of vitamin B12 occur in up to 30% of people taking long-termcanti-diabetic metformin.Deficiency does not develop if dietary intake of vitamin B12c is adequate or prophylactic B12 supplementation is given. If the deficiency is detected, metformin can be continued while the deficiency is corrected with B12 supplements..

Intramuscular injections

What are intramuscular injections?

An intramuscular injection is a technique used to deliver medication deep into the muscles. This allows the medication to be absorbed into the bloodstream quickly. You may have received an intramuscular injection at a doctors office the last time you got a vaccine, like the flu shot. In some cases, a person may also self-administer an intramuscular injection. For example, certain drugs that treat multiple sclerosis or rheumatoid arthritis may require self-injection.

What are intramuscular injections used for?

Intramuscular injections are a common practice in modern medicine. They are used to deliver drugs and vaccines. sSeveral drugs and almost all injectable vaccines are delivered this way.

Intramuscular injections are used when other types of delivery methods aren't recommended. These include:

- Oral (swallowed into the stomach)
- Intravenous (injected into the vein)
- Subcutaneous (injected into the fatty tissue just under the layer of skin)

Intramuscular injections may be used instead of intravenous injections because some drugs are irritating to veins, or because a suitable vein cant be located. It may be used instead of oral delivery because some drugs are destroyed by the digestive system when a drug is swallowed.

Intramuscular injections are absorbed faster than subcutaneous injections. This is because muscle tissue has a greater blood supply than the tissue just under the skin. Muscle tissue can also hold a larger volume of medication than subcutaneous tissue.

Intramuscular injection sites

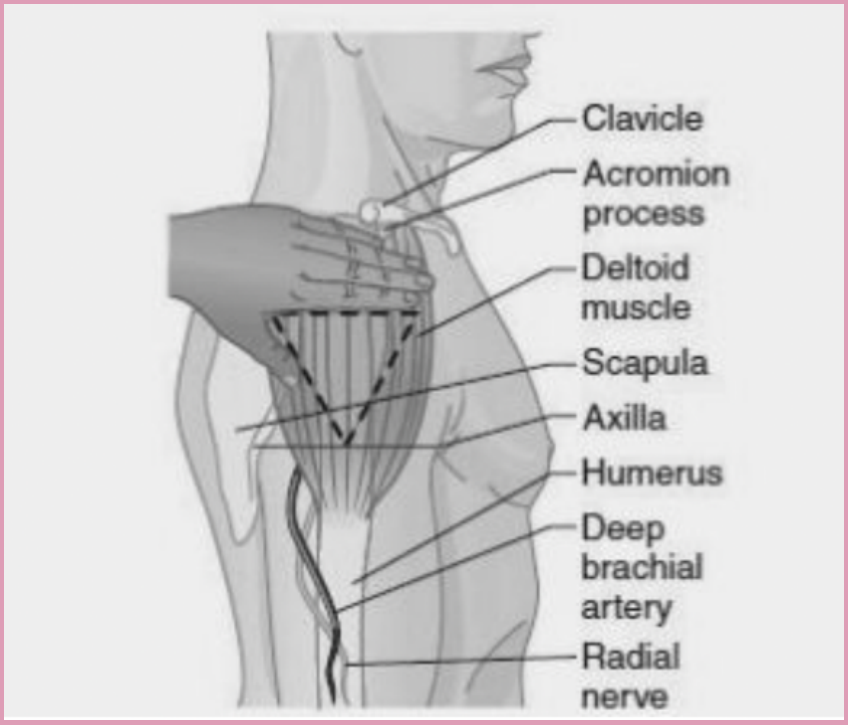
Intramuscular injections are often given in the following areas:

Deltoid muscle of the arm

The deltoid muscle is the site most typically used for vaccines. However, this site is not common for self-injection, because its small muscle mass limits the volume of medication that can be injected - typically no more than 1 millilitre, It's also difficult to use this site for self-injection. A care-giver, friend or family member can assist with injections into this muscle.

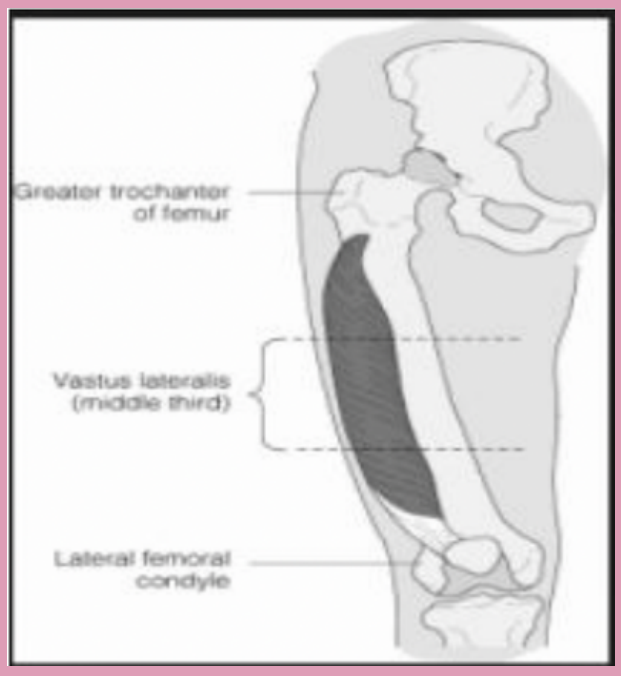
To locate this site, feel for the bone (acromion process) that's located at the top of the upper arm. The correct area to give the injection is two finger widths below the acromion process. At the bottom of the two fingers, will be an upside-down triangle.

Give the injection in the centre of the triangle.



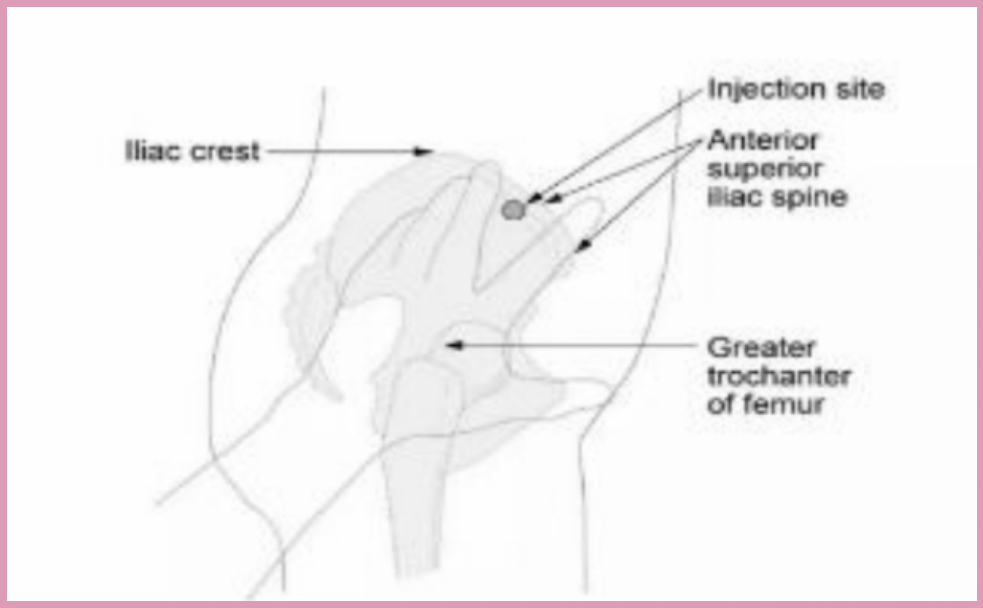
Vastus Lateralis muscle of the thigh

The thigh may be used when the other sites are not available or if you need to administer the medication on your own.
Divide the upper thigh into three equal parts. Locate the middle of these three sections.
The injection should go into the outer top portion of this section.



Ventrogluteal muscle of the hip

The ventrogluteal muscle is the safest site for adults and children older than 7 months. Its deep and not close to any major blood vessels and nerves. This site is difficult for self-injection and may require the help of a friend, family member or care-giver. Place the heel of your hand on the hip of the person receiving the injection, with the fingers pointing towards their head. Position the fingers so the thumb points towards the groin and you feel the pelvis under your pinky finger. Spread your index and middle fingers in a slight V shape and inject the needle into the middle of that V.



Dorsogluteal muscles of the buttoks

The dorsogluteal muscle of the buttocks was the site most commonly selected by healthcare providers for many years. However, due to the potential for injury to the sciatic nerve, the ventrogluteal is most often used now. This site is difficult to use for self injection and not recommended. You shouldn't use an injection site that has evidence of infection or injury. If you'll be giving the injection more than once, make sure to rotate injection sites to avoid injury or discomfort to the muscles.

How to administer an intramuscular injection

Any person who administers intramuscular injections should receive training and education on proper injection technique.
The needle size and injection site will depend on many factors. These include the age and size of the person receiving the medication and the volume and type of medication. Your doctor or pharmacist will give you specific guidelines about which needle and syringes are appropriate to administer your medication. The needle should be long enough to reach the muscle without penetrating the nerves and blood vessels underneath. Generally, beedles should be 1 inch to 1,5 inches for an adult and will be smaller for a child. They'll be 22-gauge to 25 gauge thick, noted as 22g on the packaging.
Follow these steps for a safe intramuscular injection:

1) Wash your hands

i. Wash your hands with soap and warm water to prevent potential infection. Be sure to thoroughly scrub between fingers, on the backs of hands and under fingernails. ii.The centre for disease and control and prevention (CDC) recommends lathering for 20 secs - the time it takes to sing happy birthday song 2x

2) Gather all needed supplies

i. Assemble the following supplies
ii. Needle and syringe with medication
iii. Alcohol pads
iv. Gauze
v. Sharps container to discard the used needles and syringes
vi. Bandages

3) Locate injection site

i. To isolate the muscle and target where you'll place the injection, spread the skin at injection site between two fingers. The person receiving the injection should get into a position that's comfortable, provides easy access to the location and keeps the muscles relaxed.

4) Clean injection site

i. Clean the site selected for injection with an alcohol swab and allow the skin to air dry.

5) Prepare syringe with medication

i. Remove the cap. If the vial or pen is multi dose, take notes about when the vial was first opened. The rubber stopper should be cleaned with an alcohol swab.

ii. Draw air into the syringe. Draw back the plunger to fill the syringe with air up to the dose that you'll be injecting. This was done because the vial is a vacuum and you need to add an equal amount of air to regulate the pressure. This also makes it easier to draw the medication into the syringe. Don't worry- if you forget this step, you can still get the medication out of the vial.

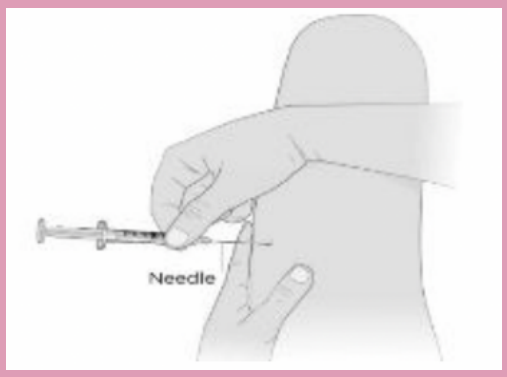
iii. insert air into the vial. Remove the cap from the needle and push the needle through the rubber stopper at the top of the vial. Inject all of the air into the vial. Be careful to not touch the needle to keep it clean.iv. Withdraw the medication. Turn the vial and syringe upside down so the needle points upward and pull back on the plunger to withdraw the correct amount of medication.

v. Remove air bubbles. Tap the syringe to push any bubbles to the top and gently depress the plunger to push air bubbles out.

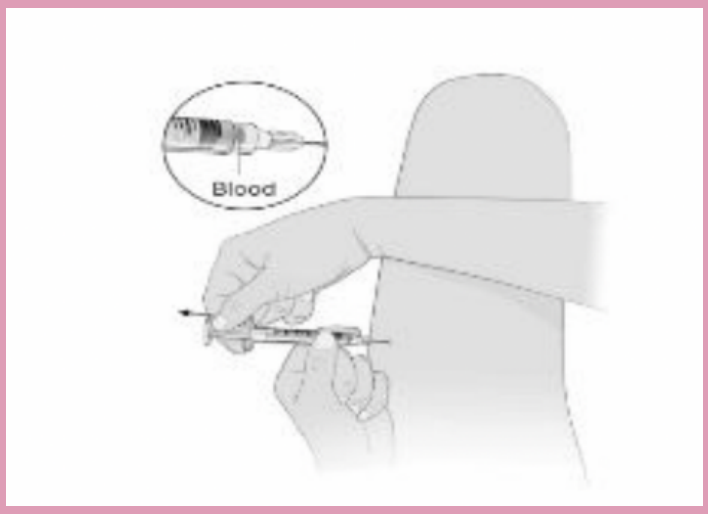
6) Self injection with a syringe

i. Insert the needle. Hold the needle like a dart and insert it into the muscles at a 90 degree angle. You should insert the needle in a quick, but controlled manner. Do not push the plunger in.

ii. Check for blood. Using the hand that's holding the skin at the injection site, pick up your index finger and thumb to stabilize the needle. Use your dominant hand - the one that did the injection - to pull back on the plunger slightly, looking for blood in the syringe. Ask your doctor if this is needed for the type of medicine you will be injecting, as its not required for all injections.



iii. If you see blood going into the syringe, it means the tip of the needle is in a blood vessel. If this happens, withdraw the needle and begin again with a new needle syringe
with medication and injection site. It's rare to have this happen.
iv. If you don't see blood going into the syringe, the needle is in the correct place and you can inject the medicine.



7) Inject the medication

i. Push the plunger slowly to inject the medication into the muscle.

8) Remove the needle

i. Withdraw the needle quickly and discard it into a puncture-resistant sharps container.
Don’t recap the needle
ii. A sharps container is a yellow container that you can purchase at any pharmacy. It's used to collect medical waste such as needles and syringes. You shouldn't put any of
these materials into the regular garbage, as needles can be hazardous to anyone who handles the rubbish.

9) Apply pressure to the injection site

i. Use a piece of gauze to apply light pressure to the injection site. You can even massage the area to help the medicine be absorbed into the muscle. Its normal to see slight
bleeding. Use a bandage if necessary.

Tips for easier injection

● To minimize possible discomfort before your injection.
● Apply ice or an over the counter topical numbing cream to the injection site before cleaning it with the alcohol pad.
● Allow the alcohol to dry completely before the injection. Otherwise, it might cause stinging.
● Warm the vial of medication by rubbing it between your hands prior to drawing the medication into the syringe
● Have someone you trust to give you the injection. Some people find it difficult to inject themselves.

What are the complications of intramuscular injections?

Its normal to experience some discomfort after an intramuscular injection, but certain symptoms may be a sign of a more serious complication. Call your doctor or healthcare provider right away if you experience:

● Severe pain at the injection site
● Tingling or numbness
● Redness, swelling or warmth at the injection site
● Drainage at the injection site
● Prolonged bleeding
● Signs of an allergic reaction, such as difficulty breathing or facial swelling

It's also normal to have some anxiety about performing or receiving an injection, especially an intramuscular injection due to the long needle. Read through the steps several times until you feel comfortable with the procedure and take your time.

Potential Health Benefits

Given the vital roles that vitamin B12 plays in your body, a deficiency can have serious health consequences. In fact, low blood levels of the vitamin have been linked to several health problems.

Brain Function

Low levels of vitamin B12 have been linked to a decline in brain function. Two recent reviews found that there may be a link between low blood levels and the development of dementia However, results have been mixed and treatment with vitamin B12 wasn't effective at improving brain function in people with normal brain function

Depression

It's been suggested that there may be a link between low vitamin B12 levels and depression. However, one review found that treating depression with vitamin B12 didn't reduce the severity of symptoms. Nevertheless, it was suggested that taking the vitamin on a long-term basis could help prevent a relapse into depression Currently, there is a lack of quality research in this area. Higher quality studies are needed to find out if there is a link between vitamin B12 and depression

Osteoporosis

Osteoporosis is a disease in which the loss of bone mass results in weaker bones and an increased risk of bone fractures. Interestingly, low blood levels of vitamin B12 have been linked with reduced bone mass Therefore, it's been suggested that taking vitamin B12 may reduce your risk of osteoporosis. However, studies have provided mixed results.

Age-Related Macular Degeneration

Age-related macular degeneration is a condition that causes you to gradually lose central vision, usually in both eyes.

In people aged 50 and over, adequate consumption of vitamin B12 is thought to be important for maintaining good vision and protecting against macular degeneration. In one large study, 5,200 women received 1,000 mcg of vitamin B12 daily, as well as other B vitamins and 7 years later, the study found a 35% lower risk of age-related macular degeneration among the women who took the supplements. Although the reduction in risk can't be attributed to vitamin B12 only, it does suggest that getting enough may be important.

Other Claims

Recently, vitamin B12 injections and infusions have become popular among healthy people who don't appear to have a deficiency. Advocates of this approach claim that regular injections can boost energy levels and help with weight loss and mood. However, there is little to no evidence to support these claims.

Contraindications

● Some medicines are not suitable for people with certain conditions, and sometimes a medicine may only be used if extra care is taken. For these reasons, before you start having hydroxocobalamin injections it is important that your doctor knows:

● If you are pregnant or breastfeeding. (Hydroxocobalamin is not known to be harmful to an unborn baby or while breastfeeding, but nevertheless you should let your doctor know about this.)

● If you have ever had an allergic reaction to a medicine.

● If you are taking any other medicines. This includes any medicines you are taking which are available to buy without a prescription, as well as herbal and complementary medicines.

Aftercare advice

● Not diet-related. If your vitamin B12 deficiency is not caused by a lack of vitamin B12 in your diet, you'll usually need to have an injection of hydroxocobalamin every 2 to 3 months for the rest of your life.

● Obtain good health eating

● No heavy lifting

● Drink plenty of water

● compress if swelling occur and seek GP advice

● Rest and no strenuous activities.

Why do aesthetics practitioners need to work with a prescriber?

Non-prescribing aesthetic practitioners need to work with a registered prescriber – or a prescribing service – in order to obtain the products they need to deliver their aesthetics treatments.

B12 is a prescription-only medication (POM). Therefore, it must be prescribed by a doctor, dentist, nurse prescriber or prescribing pharmacist who has completed training in administering Botox.

What's the best way to find a prescriber for my aesthetics business?

We would advise getting a personal recommendation, where possible. If you don't know any prescribers, try asking your peers and other medical professionals – especially those who are local to where you will be practicing. Fellow students from your training courses, or the clinical trainers themselves, may also be able to recommend registered prescribers to you.

If a non-prescriber and a prescriber choose to 'buddy up', both need to have CPD-accredited certification to show they have successfully completed a minimum of Foundation Training (or an equivalent). They also need to have the appropriate insurance coverage.

The prescriber must have one full year of experience within the field before prescribing for anyone else within aesthetics.

Alternatively, you may prefer to use a prescriber service. These organisations will send a member of their network of registered prescribers to consult with your patient and write a script, when needed.

In Great Britain you can check if a pharmacist is registered with the General Pharmaceutical Council (GPhC) by entering their name and/or registration number into the GPhC pharmacists database.

How do I work with a prescriber to order filler and botox?

Although you will be performing the actual treatment, prescribers must first carry out a face-to-face consultation with each patient prior to writing their individual script for botulinum toxin. This means that, whether you work with an individual or a prescribing service, they will need to conduct a medical consultation with each patient in person. Virtual prescriber consultations, via Zoom or Skype for example, are not permitted for B12 due to it being a POM.

How can I tell if a pharmacy is genuine?

All pharmacies in Great Britain, including online pharmacies, must be registered with the GPhC. They must also meet the specific standards required of those registered pharmacies. You can check if a British pharmacy is registered by entering their details into the searchable GPhC pharmacy database.