Policy

*Please see amendment for Pennsylvania Medicaid at the end of this CPB.

Aetna considers therapeutic phlebotomy medically necessary for any of the following indications:

- Hemochromatosis (including hereditary hemochromatosis); or
- Polycythemia vera; or
- Polycythemia* secondary to arterio-venous (A-V) fistulae; or
- Polycythemia* secondary to cor pulmonale; or
- Polycythemia* secondary to cyanotic congenital heart disease; or
- Porphyria cutanea tarda; or
- Sickle cell crisis.

* For persons with hematocrit greater than 60%.

Aetna considers therapeutic phlebotomy experimental and investigational for the following indications because its effectiveness for these indications has not been established (not an all-inclusive list).

- For use as adjunctive therapy with interferon for treatment
of chronic hepatitis C
- For the treatment of hemoglobin SC disease
- For the treatment of migraines
- For the treatment of myeloproliferative disorders without polycythemia vera
- For the treatment of progressive multiple sclerosis

**Background**

Phlebotomy (therapeutic bleeding) is a controlled removal of a large volume (usually a pint or more) of blood. It is used mainly to reduce blood volume, red cell mass and iron stores. Therapeutic phlebotomy may be indicated for hemochromatosis, polycythemia vera, porphyria cutanea tarda, and polycythemia secondary to arterio-venous fistulae, cyanotic congenital heart disease or cor pulmonale.

Therapeutic phlebotomy is used to remove excess iron and maintain low normal body iron stores in patients with hemochromatosis. According to guidelines from the Hemochromatosis Management Working Group (Barton et al, 1998), therapeutic phlebotomy should be initiated in men with serum ferritin levels of 300 ug/L or more and in women with serum ferritin levels of 200 ug/L or more, regardless of the presence or absence of symptoms. Typically, therapeutic phlebotomy consists of (i) removal of 1 unit (450 to 500 ml) of blood weekly until the serum ferritin level is 10 to 20 ug/L, and (ii) maintenance of the serum ferritin level at 50 ug/L or less thereafter by periodic removal of blood. Hyperferritinemia attributable to iron overload is resolved by therapeutic phlebotomy. When applied before iron overload becomes severe, this treatment also prevents complications of iron overload, including hepatic cirrhosis, primary liver cancer, diabetes mellitus, hypogonadotrophic hypogonadism, joint disease, and cardiomyopathy. In patients with established iron overload disease, weakness, fatigue, increased hepatic enzyme concentrations, right upper quadrant pain, and hyperpigmentation are often substantially alleviated by therapeutic phlebotomy.
Serum iron and ferritin concentrations are frequently elevated in patients with chronic viral hepatitis. Pilot studies suggested that HCV-infected patients with elevated concentrations of iron in the blood and liver are less likely to respond to interferon, and that the response could be enhanced with iron reduction. However, randomized controlled clinical trials of therapeutic phlebotomy for HCV treatment have not found significant improvements in sustained virologic response with phlebotomy plus interferon (IFN) compared to IFN alone (Fonatana et al, 2000; Di Bisceglie et al, 2000).

In a meta-analysis of randomized controlled trials (RCTs), Desai and colleagues (2008) compared phlebotomy and IFN to IFN alone for the treatment of chronic hepatitis C (CHC). The Medline database and Cochrane registry of controlled trials were searched using the key words "phlebotomy" and "treatment of hepatitis C." Reference lists of review articles discussing the interaction between iron and CHC, and prospective RCTs comparing phlebotomy plus IFN therapy to IFN alone were searched to identify additional RCTs that compared phlebotomy plus IFN to IFN alone. Peto odds ratios with their 95 % confidence intervals (CI) and Forrest plots were generated for each variable to assess the relationships among the studies that had provided that information. Statistical analysis was performed using Comprehensive Meta-Analysis version 2.0. A total of 6 prospective RCTs were identified: all used sustained viral response (SVR) as an endpoint. The 3 largest RCTs excluded patients with cirrhosis. Two RCTs specifically included only patients with either high ferritin or high hepatic iron content. Interferon treatment regimes varied. Length of treatment varied between 6 and 12 months. The phlebotomy plus IFN group and the IFN group did not differ with respect to the percentage of patients with cirrhosis or genotype 1. Sustained viral response was attained in 50/182 (27 %) patients in the phlebotomy plus IFN group, compared to 22/185 (12 %) patients in the IFN group. Peto odds ratio for SVR in phlebotomy plus IFN group was 2.7; 95 % CI: 1.6 to 4.5, p < 0.0001. All 5 RCTs published in manuscript form showed a trend towards a benefit from the phlebotomy plus IFN in
attaining SVR, and the results of the meta-analysis were not dependent on any single RCT, since excluding any single RCT did not change the results. The authors stated that phlebotomy appeared to enhance the efficacy of non-pegylated IFN monotherapy for CHC, but more research was required to confirm this. Problems associated with the limited volume of data and clinical and methodological heterogeneity between the studies were acknowledged and addressed by the investigators in the discussion section of the review. The doubtful applicability of this evidence to pegylated IFN was also highlighted. Moreover, the authors stated that confirmation of this will require RCT with detailed pre-treatment iron studies and appropriately powered to demonstrate a statistically significant benefit. The authors stated that adequately powered RCTs with detailed pre-treatment iron studies should be considered to evaluate phlebotomy as an adjunct to pegylated IFN, with or without ribavirin. As a priority, they recommended research among selected genotype one patients unable to tolerate ribavirin.

Guidelines from the American Gastroenterological Association (Dienstag and McHutchison, 2006) on management of hepatitis C concluded that clinical trials have failed to demonstrate the efficacy of phlebotomy in patients with chronic HCV infection, and that phlebotomy cannot currently be recommended as a treatment for HCV infection.

In a review on evidence-based approach for the treatment of adults with sickle cell disease, Lottenberg and Hassell (2005) noted that reports and case series indicated that repeated phlebotomy to lower the hemoglobin (Hb) level and induce iron deficiency can reduce the frequency of painful episodes in selected patients with high steady state Hb levels.

Bouchair et al (2000) reported the findings of sickle cell disease patients who suffered from frequent painful crises and were submitted to phlebotomies in order to reduce hospitalization days due to pain. These patients had an Hb level equal to or above 9.5 g/dL. A total of 7 sickle cell disease patients (4 sickle
cell anemia, 3 sickle Hb C disease), aged 4 to 24 years, were submitted to sequential phlebotomies during periods from 18 months to 4 years. The number of hospitalization days for crises was considered. The volumes and frequencies of phlebotomies were adjusted according to the patients ages, the Hb concentrations and the serum ferritin levels. A total of 144 hospitalization days were recorded in the 7 patients in the year preceding the treatment. During the study period, the annual numbers of hospitalization days were respectively 20, 5, 6 and 1. Mean Hb concentration was 10.7 g/dL before phlebotomies and 8.8 to 9.2 g/dL during the 4 years of treatment. Mean corpuscular volume, mean corpuscular Hb concentration and serum ferritin were also reduced. The volume of phlebotomies was 116 to 390 ml/kg/year according to the patients. The striking decrease of the number of hospitalization days for all the patients suggests a closed relationship between therapy and clinical improvement. The mechanism of this effect is probably multi-factorial: (i) the concentration of Hb level is known to influence the blood viscosity and its decrease always improved rheology in sickle cell disease patients; (ii) the mean corpuscular Hb concentration is a critical factor concerning the HbS molecule polymerization in sickle cell disease, and its slight reduction may have an important biological effect. The authors observed these two biological modifications in their patients and suggested that they mediate the clinical effects. The iron deficiency induced by phlebotomies has no evident deleterious consequence either on height and weight in the children or on intellectual performance in any patients.

Rombos and colleagues (2002) noted that sickle cell disease patients who acquire iron deficiency may experience a degree of amelioration from painful crises in terms of frequency, severity, and duration. This observation prompted these researchers to identify the potential utility of iron load reduction in the management of this disease. A total of 13 sickle cell patients not ameliorated by conventional treatment entered a weekly venesection protocol (phlebotomy). Hematological values and painful crises of all degrees of severity were recorded and compared to those of
the last 12 months before venesection for each case separately (historical controls). A decrease was noted in the frequency and intensity of several types of painful crises. Reduction of iron load by venesection seems to be a simple, safe, side-effect-free, and efficient way of preventing and ameliorating to a large extent painful crises in sickle cell disease.

Markham et al (2003) stated that marked variability is a keynote in the disease course of patients with Hb SC (Hb SC) and hemoglobin S/beta(+)thalassemia (Hb S/beta(+)‐thal), with some patients having a frequency of complications and painful episodes similar to patients with homozygous sickle cell (Hb SS) disease. One possible explanation is that the higher hematocrit in these syndromes may contribute to an increase in blood viscosity, leading to vaso-occlusive pain episodes as well as an increased incidence of thrombo-embolic complications and retinopathy. These investigators presented a patient with Hb SC disease with an excellent baseline functional status who developed splenic infarction at a high altitude. Following splenectomy, the patient developed a sustained increase in hematocrit, an increase in the frequency of painful episodes, as well as new-onset dizziness and malaise. The authors initiated a therapeutic phlebotomy program in order to lower the hematocrit to pre-splenectomy values, as well as to induce iron deficiency. Repeated phlebotomy resulted in a dramatic decrease in symptoms. This patient no longer requires narcotic analgesics for pain, has resolution of constitutional symptoms, and has not required further hospitalizations for vaso-occlusive pain crises. The correlation between symptoms and hematocrit levels supports the importance of blood viscosity in contributing to this patient's symptoms. A trial of phlebotomy to reduce viscosity in patients with higher hematocrit values should be considered as an intervention for symptomatic patients with sickle cell disease.

The American Association for the Study of Liver Diseases' clinical practice guideline on "Diagnosis and management of hemochromatosis" (Bacon et al, 2011) provided the following
recommendations:

- Patients with hemochromatosis and iron overload should undergo therapeutic phlebotomy weekly (as tolerated). Target levels of phlebotomy should be a ferritin level of 50 to 100 µg/L.
- In the absence of indicators suggestive of significant liver disease (ALT, AST elevation), C282Y homozygotes who have an elevated ferritin (but less than 1,000 µg/L) should proceed to phlebotomy without a liver biopsy.
- Patients with end-organ damage due to iron overload should undergo regular phlebotomy to the same endpoints as indicated above.
- During treatment for hereditary hemochromatosis, dietary adjustments are unnecessary. Vitamin C supplements and iron supplements should be avoided.
- Patients with hemochromatosis and iron overload should be monitored for re-accumulation of iron and undergo maintenance phlebotomy. Target levels of phlebotomy should be a ferritin level of 50 to 100 µg/L.
- The guideline developers recommend treatment by phlebotomy of patients with non-HFE iron overload who have an elevated hepatic iron concentration.

Barbui and colleagues (2011) presented a review of critical concepts and produced recommendations on the management of Philadelphia-negative classical myeloproliferative neoplasms, including monitoring, response definition, first- and second-line therapy, and therapy for special issues. Key questions were selected according the criterion of clinical relevance. Statements were produced using a Delphi process, and 2 consensus conferences involving a panel of 21 experts appointed by the European LeukemiaNet (ELN) were convened. Patients with polycythemia vera (PV) and essential thrombocythemia (ET) should be defined as high-risk if age is greater than 60 years or there is a history of previous thrombosis. Risk stratification in primary myelofibrosis (PMF) should start with the International Prognostic Scoring System (IPSS) for newly diagnosed patients and dynamic IPSS for
patients being seen during their disease course, with the addition of cytogenetics evaluation and transfusion status. High-risk patients with PV should be managed with phlebotomy, low-dose aspirin, and cytoreduction, with either hydroxyurea or interferon at any age. High-risk patients with ET should be managed with cytoreduction, using hydroxyurea at any age. Monitoring response in PV and ET should use the ELN clinico-hematologic criteria. Corticosteroids, androgens, erythropoiesis-stimulating agents, and immunomodulators are recommended to treat anemia of PMF, whereas hydroxyurea is the first-line treatment of PMF-associated splenomegaly. Indications for splenectomy include symptomatic portal hypertension, drug-refractory painful splenomegaly, and frequent red blood cells transfusions. The risk of allogeneic stem-cell transplantation-related complications is justified in transplantation-eligible patients whose median survival time is expected to be less than 5 years.

Tefferi (2012) stated that PV and ET are myelo-proliferative neoplasms (MPN) primarily characterized by erythrocytosis and thrombocytosis, respectively. Other disease features include leukocytosis, splenomegaly, thrombo-hemorrhagic complications, vasomotor disturbances, pruritus, and a small risk of disease progression into acute myeloid leukemia or myelofibrosis. Almost all patients with PV harbor a JAK2 mutation. When PV is suspected, the presence of a JAK2 mutation confirms the diagnosis and its absence, combined with normal or increased serum erythropoietin level, excludes the diagnosis. Differential diagnosis of ET had to include chronic myelogenous leukemia and pre-fibrotic myelofibrosis. A JAK2 mutation is found in approximately 60% of patients with ET. Current risk stratification in PV and ET is designed to estimate the likelihood of thrombotic complications: high-risk is defined by the presence of age greater than 60 years or presence of thrombosis history; low-risk is defined by the absence of both of these 2 risk factors. Presence of extreme thrombocytosis (platelet count greater than $1,000 \times 10^9$/L) might be associated with acquired von Willebrand syndrome (AvWS) and, therefore, risk of bleeding. Risk factors for
shortened survival in both PV and ET include advanced age, leukocytosis, and history of thrombosis. Survival is near-normal in ET and reasonably long in PV. The 10-year risk of leukemic/fibrotic transformation is less than 1%/1% in ET and less than 3%/10% in PV. In contrast, the risk of thrombosis exceeds 20%. The main goal of therapy is therefore to prevent thrombo-hemorrhagic complications and this is effectively and safely accomplished by the use of low-dose aspirin (PV and ET), phlebotomy (PV), and hydroxyurea (high-risk PV and ET). Treatment with busulfan or interferon-α is usually effective in hydroxyurea failures. Screening for clinically significant AvWS is recommended before administrating aspirin in the presence of extreme thrombocytosis.

Lengfelder (2013) presented an overview on relevant topics of pathogenesis and diagnosis of PV. The presently available treatment options in PV were discussed and recommendations for the clinical management were given. The JAK2V617F mutation, a point mutation in the tyrosine kinase gene JAK2 (Janus Kinase 2), has emerged as a central feature in the pathogenesis of MPN. Subsequently, the identification of several other mutated genes in MPN has shown that the pathogenesis is complex and that the JAK2V617F mutation is a critical, but not the only step leading to the uncontrolled proliferation in MPN including PV. The diagnostic criteria of PV have been revised in 2008 and include the JAK2V617F mutation as one of the 2 major criteria of the disease. This molecular diagnostic marker proves the clonality and facilitates the diagnosis of early and uncertain cases that remained sometimes undiagnosed in the past. Main treatment aims are the reduction of thromboembolic events and the minimization of the risk of myelofibrosis and of acute leukemia. The authors concluded that PV patients with low-risk of vascular complications should be treated with phlebotomy and low-dose acetylsalicylic acid. High-risk patients should receive cytoreductive therapy with hydroxyurea or interferon alpha. Studies with JAK inhibitors are presently ongoing.

An UpToDate review on “Prognosis and treatment of
polycythemia vera” (Tefferi, 2013) states that “In subjects without active thrombosis and those not at risk for thrombosis (i.e., age of less than 60, no prior thrombosis), we recommend that the hematocrit be kept within the normal range via the use of serial phlebotomy, rather than by the use of myelosuppressive agents (Grade 1A). Optimal control is to keep the hematocrit below 45 %in men and 42 % in women. Since phlebotomy is effective in controlling PV by producing a state of relative or absolute iron deficiency, iron supplementation should not be given. For patients at high risk for thrombosis (i.e., age of greater than 60, prior thrombosis), we recommend that treatment with phlebotomy be supplemented with the use of a myelosuppressive agent. (Grade 1B). For this purpose we prefer the use of hydroxyurea rather than an alkylating agent, radioactive 32P, or interferon alpha. If not otherwise contraindicated because of a history of major bleeding or intolerance, we suggest that aspirin be given to all patients (Grade 2C). The appropriate dose is 75 to 100 mg/day. Treatment with higher doses should be avoided”.

In a pilot study, Creange et al (2013) evaluated the concept that iron depletion (ID) induced by blood-letting and followed by recombinant human erythropoietin (rhEPO) administration could be a therapeutic strategy in progressive multiple sclerosis (PMS) and that it could be assessed by neurophysiological measurements. In 4 patients with PMS, blood-letting was performed until ID was induced, and then rhEPO was administered (300 UI/kg/week). The changes induced by the treatment were assessed by clinical scores, biological tests, and neurophysiological study of cortical excitability using transcranial magnetic stimulation techniques. The treatment was well-tolerated except for muscle cramps and 1 popliteal vein thrombosis in a patient confined to chair. Iron depletion was obtained within 28 weeks and was associated with endogenous production of EPO. No blood-letting was further required during a 6-month period after introduction of rhEPO. At the end of the follow-up (up to 1 year), fatigue and walking capacities tended to improve in 2 patients. Neurophysiological changes were characterized by an increased cortical excitability,
including a decrease of motor thresholds and an enhancement of intra-cortical facilitation and cerebello-thalamo-cortical inhibition. The authors concluded that the combined ID-rhEPO therapy could authorize a prolonged administration of rhEPO in PMS patients, able to modify cortical excitability of the glutamatergic and gabaergic circuits. Moreover, they stated that these preliminary data are encouraging to design a larger, controlled trial to assess the value of such a strategy to improve functional symptoms in PMS patients, and maybe to prevent axonal degeneration.

In a Cochrane review, Wang and Dwan (2013) evaluated risks and benefits of chronic blood transfusion regimens in people with sickle cell disease to prevent first stroke or recurrences. These investigators searched the Cochrane Cystic Fibrosis and Genetic Disorders Group Trials Register, comprising references identified from comprehensive electronic database searches and hand-searches of relevant journals and conference proceedings. Date of the latest search of the Group’s Haemoglobinopathies Trials Register was January 28, 2013. Randomized and quasi-randomized controlled trials comparing blood transfusion as prophylaxis for stroke in people with sickle cell disease to alternative or no treatment were selected for analysis. Both authors independently assessed the risk of bias of the included trials and extracted data. Searches identified 3 eligible randomized trials (n = 342). The first 2 trials addressed the use of chronic transfusion to prevent primary stroke; the third utilized the drug hydroxycarbamide (hydroxyurea) and phlebotomy to prevent both recurrent (secondary) stroke and iron over-load in patients who had already experienced an initial stroke. In the first trial (STOP) a chronic transfusion regimen for maintaining sickle hemoglobin lower than 30% was compared with standard care in 130 children with sickle cell disease judged (through transcranial Doppler ultrasonography) as high-risk for first stroke. During the trial, 11 children in the standard care group suffered a stroke compared to 1 in the transfusion group, odds ratio [OR] of 0.08 (95% CI: 0.01 to 0.66). This meant the trial was terminated early. The transfusion group had a high complications rate, including iron
over-load, allo-immunization, and transfusion reactions. The second trial (STOP II) investigated risk of stroke when transfusion was stopped after at least 30 months in this population. The trial closed early due to a significant difference in risk of stroke between participants who stopped transfusion and those who continued as measured by re-occurrence of abnormal velocities on Doppler examination or the occurrence of overt stroke in the group that stopped transfusion. The third trial (SWiITCH) was a non-inferiority trial comparing transfusion and iron chelation (standard management) with hydroxyurea and phlebotomy (alternative treatment) with the combination end-point of prevention of stroke recurrence and reduction of iron over-load. This trial was stopped early after enrolment and follow-up of 133 children because of analysis showing futility in reaching the composite primary end-point. The stroke rate (7 strokes on hydroxyurea and phlebotomy, none on transfusion and chelation, OR of 16.49 (95 % CI: 0.92 to 294.84)) was within the non-inferiority margin, but the liver iron content was not better in the alternative arm. The authors concluded that the STOP trial demonstrated a significantly reduced risk of stroke in participants with abnormal transcranial Doppler ultrasonography velocities receiving regular blood transfusions. The follow-up trial (STOP 2) indicated that individuals may revert to former risk status if transfusion is discontinued. The degree of risk must be balanced against the burden of chronic transfusions. The combination of hydroxyurea and phlebotomy is not as effective as "standard" transfusion and chelation in preventing secondary stroke and iron over-load. Moreover, they stated that ongoing multi-center trials are investigating the use of chronic transfusion to prevent silent infarcts, the use of hydroxyurea as an alternative to transfusion in children with abnormal transcranial Doppler ultrasonography velocities, and the use of hydroxyurea to prevent conversion of transcranial Doppler ultrasonography velocities from conditional (borderline) to abnormal values.

Du and colleagues (2014) evaluated the therapeutic effect of pricking blood therapy for migraine. These investigators searched all the original papers about pricking blood therapy
for migraine in common databases as the Chinese National Knowledge Infrastructure Database (CNKI), Chinese Biomedical Library (CBM), Ovid, Science Direct, Socolar, and Sci Finder (1949 to 2012), Wanfang Data (1998 to 2012) and Foreign Medical Journal Service (FMJS, 1990 to 2012). The original articles were searched in accordance with a pre-defined standards (simple pricking blood treatment, or the pricking blood therapy was the principal approach), while those about other diseases (such as cerebrovascular disease, cervical spondylosis, etc.) evoked migraine, pricking blood used as a complementary therapy, case report, specialists' experience summary, reviews, surveys, news articles, animal studies were excluded. Then, a Meta-analysis was made by software Review Manager 5.1. A total of 11 clinical trial papers involving 826 cases of migraine were included in the present paper; 3 of them were high-quality researches, and the other 8 were low quality researches. Results of meta-analysis indicated that the therapeutic effect of the pricking blood therapy was significantly superior to that of non-bleeding therapies in relieving migraine [OR = 6.23, 95 % CI: 4.03 to 9.63, Z = 8.24, p < 1.1 \times 10^{-01}]. However, the poor symmetry of funnel plot suggested a risk of bias. The authors concluded that the pricking blood therapy is effective for relieving migraine, but larger sample clinical trials, particularly RCTs are definitely needed for confirming the conclusion.

Furthermore, UpToDate reviews on “Preventive treatment of migraine in adults” (Bajwa and Smith, 2015) and “Chronic migraine” (Garza and Schwedt, 2015) do not mention phlebotomy as a therapeutic option.

**Hemoglobin SC Disease:**

Summarell and Sheehan (2016) stated that hydroxyurea is an excellent therapeutic agent for the pharmacological induction of fetal hemoglobin (HbF) in patients with sickle cell disease (SCD). However, all completed clinical trials of hydroxyurea have excluded patients with hemoglobin SC (HbSC) disease; HbSC differs significantly in pathophysiology from HbSS, as HbC does
not sickle, but instead causes cellular dehydration which potentiates sickling of HbS. Many severely affected HbSC patients have been placed on hydroxyurea on a case-by-case basis, but there are no large scale prospective data on safety or effectiveness of hydroxyurea in this subset of patients with SCD. These investigators reported a case series of 14 pediatric patients with HbSC treated to maximum tolerated dose (MTD) with hydroxyurea. Those who failed to show clinical improvement after at least 6 months at MTD were offered phlebotomy in addition to hydroxyurea; 5 out of 11 patients with HbSC who achieved MTD failed to demonstrate clinical improvement on hydroxyurea. Of the 4 placed on dual hydroxyurea and phlebotomy therapy, all showed at least partial clinical improvement. Percent dense red blood cells (%DRBC) were measured via an ADVIA hematology analyzer. A marked rise in percent dense cells preceded clinical complications in 3 patients. Dual therapy with hydroxyurea and phlebotomy may be an effective approach to patients with HbSC that do not experience improvement with hydroxyurea alone. Monitoring of %DRBC may predict adverse events and aid in evaluating hydroxyurea compliance. The authors concluded that large scale clinical trials are needed to evaluate the safety and effectiveness of hydroxyurea and hydroxyurea with phlebotomy in patients with HbSC disease.

<table>
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<tr>
<th>CPT Codes / HCPCS Codes / ICD-10 Codes</th>
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<tr>
<td>Information in the [brackets] below has been added for clarification purposes. Codes requiring a 7th character are represented by &quot;+&quot;:</td>
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<td>ICD-10 codes will become effective as of October 1, 2015:</td>
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<tr>
<td>CPT codes covered if selection criteria are met:</td>
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<tr>
<td>99195 Phlebotomy, therapeutic (separate procedure)</td>
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<td>Other CPT codes related to the CPB:</td>
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<td>36415 Collection of venous blood by venipuncture</td>
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<td>Other HCPCS codes related to the CPB:</td>
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**ICD-10 codes covered if selection criteria are met:**

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<tr>
<td>C94.00 -</td>
<td>Acute erythoid leukemia</td>
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<td>C94.01</td>
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<td>D45</td>
<td>Polycythemia vera</td>
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<td>D57.00 -</td>
<td>HB-SS disease with crisis</td>
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<td>D57.02</td>
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<td>D57.819</td>
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<td>D75.0</td>
<td>Familial erythrocytosis</td>
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D75.1  Secondary polycythemia  
E80.1  Porphyria cutanea tarda  
E83.110 - E83.119  Hemochromatosis  
P61.1  Polycythemia neonatorum  

**ICD-10 codes not covered for indications listed in the CPB (not all-inclusive):**

<table>
<thead>
<tr>
<th>Code</th>
<th>Description</th>
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<td>B18.2</td>
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<td>C92.10 - C92.22</td>
<td>Chronic myeloid leukemia, BCR/ABL-positive and negative</td>
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<td>Essential (hemorrhagic) throbocythemia [unless with Polycythemia vera]</td>
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<td>D75.81</td>
<td>Myelofibrosis [unless with Polycythemia vera]</td>
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<td>G35</td>
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<tr>
<td>K73.9</td>
<td>Chronic hepatitis, unspecified</td>
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**The above policy is based on the following references:**


44. Tefferi A. Prognosis and treatment of polycythemia vera. Last reviewed June 2013. UpToDate Inc., Waltham, MA.
AETNA BETTER HEALTH® OF PENNSYLVANIA

Amendment to
Aetna Clinical Policy Bulletin Number: 0652
Therapeutic Phlebotomy

The Pennsylvania Medical Assistance Program considers therapeutic phlebotomy to be medically necessary for the treatment of hemoglobin SC disease.